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PHYSIOLOGIC CHANGES FOLLOWING OBSTRUCTION OF THE SUPERIOR VENA CAVA

REPORT OF A CLINICAL CASE AND EXPERIMENTAL STUDIES

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IN THE course of dissecting the right pulmonary veins within the pericardium during a pneumonectomy for bronchiogenic carcinoma one of us (F. J. J.) was disturbed to find, upon completion of the dissection, that he had mistaken the intrapericardial pulmonary venous trunk (found as a frequent variation on the right) for the inferior pulmonary vein and had then inadvertently divided the superior vena cava. It was estimated that the superior vena cava had been obstructed for approximately fifteen minutes before the true state of affairs was recognized and it required forty-two minutes more to effect an anastomosis of the cava with the auricle. The azygos vein had been divided early in the course of the operation. Surprisingly enough the patient tolerated this physiologic insult quite well (death thirty hours later could be certainly attributed to respiratory functional inadequacy). We were unable to locate a report of a similar surgical misadventure although there are many reports of well-tolerated, gradual superior vena cava obstruction resulting from inflammatory or neoplastic disease.

Carlson (1934) found that obstruction of the superior vena cava is well tolerated by the dog above the azygos vein. Recent experimental observations by Andreasen and Watson (1952) demonstrated the ability of dogs to survive occlusion of the superior and inferior venae cavae for ten minutes if the azygos vein is intact. However, Carlson's two dogs with obstruction below the azygos died "in a few minutes." Since these earlier observations were at such variance

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with the behavior of our patient, studies were undertaken in the laboratory to clarify (1) the physiologic pressure changes resulting from acute occlusion of the superior vena cava and following its release, (2) the ability of the animal to survive permanent acute occlusion of the superior vena cava below the azygos vein, and (3) the role of the azygos vein in collateral return flow of venous blood to the heart.

CASE REPORT

R. H. P., a 59-year-old man, was admitted to the hospital, Jan. 27, 1953. Active pulmonary tuberculosis had been diagnosed in March, 1952, based on roentgenograms of the chest and the finding of tubercle bacilli in concentrate sputum smears. In 1918 orchidectomy had been performed for right tuberculous epididymitis, and in 1924 the left testis was removed for tuberculous disease. Right nephrectomy became necessary for tuberculous pyelonephritis in 1951, and similar involvement of the left kidney was suspected in March, 1952.

The patient was chronically ill, with physical evidence of marked pulmonary emphysema. Roentgenograms of the chest (available in series from October, 1948) revealed a fibrotic type of infiltration in the right apex which had increased moderately, especially in the upper third of the right lung, with some increased radiopacity in the hilar region. Routine laboratory studies were within normal limits. The blood sedimentation rate (corrected, Wintrobe) was 31 mm. per hour. Two acid-fast bacilli were found in the sputum on the week of admission, but cultures of the sputum for tubercle bacilli were reported positive. Pyelograms and urine studies failed to confirm the suspected tuberculosis in the remaining kidney. Bronchoscopy (June 4, 1952) revealed a partial obstruction of the right main bronchus by an irregular membranous structure which bled readily, and from which a biopsy was reported as showing squamous-cell carcinoma. Studies of the pulmonary function revealed a maximum breathing capacity of 97 L. which was 72.5 per cent of the predicted maximum for this patient. Fifty-six per cent of the oxygen consumption was on the right, 52.5 per cent of the ventilation. In spite of this extremely poor pulmonary reserve, in view of the finding of carcinoma, pneumonectomy was advised and accepted by the patient and his family after full appraisal of the great risk entailed.

Operation.—With the patient in the face-down position, the right hemithorax was entered through the bed of the resected fifth rib. There were numerous firm adhesions about the apex of the upper lobe which contained extensive palpable lesions compatible with caseonodular and fibrous tuberculosis. Although large lymph nodes were felt about the hilus, the area was not fixed and it was apparent that we were dealing with a situation that was technically operable.

Complete adherence of the apex made it necessary to free the lobe by removing a portion of the thick parietal pleural peel in the endothoracic fascial plane. The mediastinal dissection was begun by exposing the innominate artery and vein and dividing the vagus nerve just beyond the recurrent laryngeal branch superiorly. The lymphatic structures, along with the phrenic and vagus nerves, were stripped from the lateral aspects of the trachea, esophagus, and aorta. The azygos vein was divided between ligatures and used as a tag to rotate the structures of the mediastinum outward. Two large paraesophageal nodes lateral to the mid-esophagus were removed separately from the main block of tissue and the esophagus was dissected free of its surrounding tissue throughout its entire length.

There were large, firm lymph nodes lying superior to the right main-stem bronchus between it and the superior vena cava, and the nodes at the tracheal bifurcation were also large and firm, but not adherent. These were suspected strongly of being involved with carcinoma but it was impossible to tell that their enlargement was not the residuum of the old tuberculosis. The bronchus was isolated, divided, and closed with interrupted sutures of 0000 silk.

The left main bronchus was then dissected down to the level of the left inferior pulmonary vein. The right inferior pulmonary ligament was incised at its base at a point approximately 2 cm. inferior to the right inferior pulmonary vein. Here the pericardium

was entered, removing a large window of pericardium. The inferior pulmonary vein was then circumscribed and divided between ligatures of strong silk. The pericardium was dissected away from the inferior and posterior aspects of the pulmonary artery. What was taken to be the superior pulmonary vein was then circumscribed and divided between ligatures. The pulmonary artery was cleared of its surrounding pericardium and areolar tissue, ligated, and the proximal ligature bolstered by a suture ligature. The dissection was continued along the posterior and lateral aspects of the superior vena cava with difficulty because of the large adherent lymph nodes.

When this dissection had finally been accomplished and the lung removed, it became apparent that the venous conus carrying both the superior and inferior pulmonary veins into the auricle had been mistaken for the inferior pulmonary vein and ligated, and the superior vena cava, mistakenly identified as the superior pulmonary vein, had been ligated and divided. At this point it became obvious that the auricle was filling poorly and a pulse was not obtainable in the upper extremities by the anesthetist. Immediate restoration of superior vena caval continuity became imperative.

The anastomosis presented several technical problems. Proper clamps were not available with which to secure the cut end of vena cava entering the auricle. It was necessary to use a rubber covered clamp superiorly, placing it as high as possible where the right and left major venous trunks join to form the cava. In the absence of a proper clamp a ligature was placed around the auricular wall, after tenting the auricle to increase the space for it, and the original ligature was removed. It was then possible to bring the two ends of the superior vena cava together and to achieve a satisfactory anastomosis. Before completing the lateral quarter of the anterior suture line the clamp on the superior vena cava was opened allowing the clots to be washed out. There were several clots, followed by free bleeding, and the caval anastomosis was completed. The proximal clamp was then removed slowly. At the same time the anesthetist compressed the carotid vessels to minimize the possibility of cerebral embolization. With the elimination of superior caval obstruction there was prompt restoration of normal auricular filling followed by an immediate return of blood pressure and pulse in the upper extremities, the systolic pressure reaching 120 mm. Hg within a period of four to five minutes. The heart rate had at all times been below 100 and the cardiac thrust had been surprisingly good.

The anterior mediastinal nodes were then removed, completing the dissection. The chest wall was then rapidly closed in layers. At the completion of the operative procedure the patient was in very satisfactory condition. Whole blood (2,500 c.c.) had been used to replace the weighed blood loss of 1900 Gm. The procedure required approximately two hours and ten minutes.

The patient's return to consciousness was prompt and the first postoperative hours were surprisingly uncomplicated. During the night considerable difficulty was encountered in clearing the tracheobronchial tree of secretions, and as the morning approached he became markedly dyspneic. Twenty-three hours after operation there was a rapid deterioration of his condition, with signs of shock. It was impossible to obtain pulse or blood pressure in the arms, although the femoral pulses were fairly strong and slow. There was slight cyanosis and a roentgenogram of the chest showed considerable fluid in the right hemithorax. In spite of the use of necessary supportive measures to combat shock and improve respiratory exchange the patient's condition became increasingly grave. There was a progressive rise in respiratory rate and he expired approximately thirty hours postoperatively from pulmonary insufficiency.

Examination of the resected specimen* revealed a squamous-cell carcinoma originating in the right main bronchus, occluding the right upper lobe bronchus and extending to and through the orifices of the segmental bronchi. A single peribronchial lymph node contained metastatic carcinoma and a submucosal infiltration of neoplastic cells was found at the site of bronchial transection. Active caseonodular tuberculosis involved all segments

*Post-mortem examination and histologic diagnoses were reported by Dr. James G. Bennett, Pathologist to Firland Sanatorium, Seattle, Wash.

of the right lung and there was extensive tuberculosis of the mediastinal lymph nodes. The upper lobe was the site of extensive bronchiectasis with local ulceration of the bronchial mucosa and extensive chronic inflammatory changes throughout the upper lobe parenchyma.

At necropsy the anastomosis of the superior vena cava was intact and patent. Except for some excess of sanguinoserous fluid in the right hemithorax there was no demonstrable anatomic cause for death. Clinically the patient showed signs and symptoms of progressive anoxia resulting from the respiratory insufficiency, consequent to a functional pulmonary reserve inadequate to sustain him through the early postoperative period.

The period of acute superior vena cava obstruction in this patient lasted slightly less than one hour but was tolerated surprisingly well when viewed in the light of Carlson's experimental data. The problem of temporary superior vena cava obstruction, with perhaps graft replacement, may become increasingly more frequent as radical intrathoracic procedures for carcinoma become more commonplace. Experimental studies were made to determine some of the factors involved in acute temporary and permanent superior vena cava obstruction.

EXPERIMENTAL DATA

Methods.—Mongrel dogs weighing an average of 10 kilograms were used. Two groups of experiments were carried out: dogs with acute permanent superior vena caval obstruction, and dogs with acute temporary superior vena caval obstruction. In the latter group pressure recordings were obtained from the proximal superior vena cava, the right auricle, and the aorta before, during, and immediately after the obstruction of superior caval blood flow. A simultaneous electrocardiographic recording (Lead II) was obtained by using a four channel Sanborn Poly-Viso recording apparatus.

All operative procedures were performed under aseptic conditions and Nembutal anesthesia supplemented by positive pressure endotracheal ether-oxygen. The right pleural cavity was entered through the fourth intercostal space. The superior vena cava was exposed from the azygos vein to the right auricle. In six animals complete and permanent obstruction of the superior vena cava was obtained by application of two Potts coarctation clamps on the cava between the azygos vein and the right auricle, dividing the superior vena cava between these clamps and closing the cut ends by double rows of 00000 silk suture on an atraumatic needle.

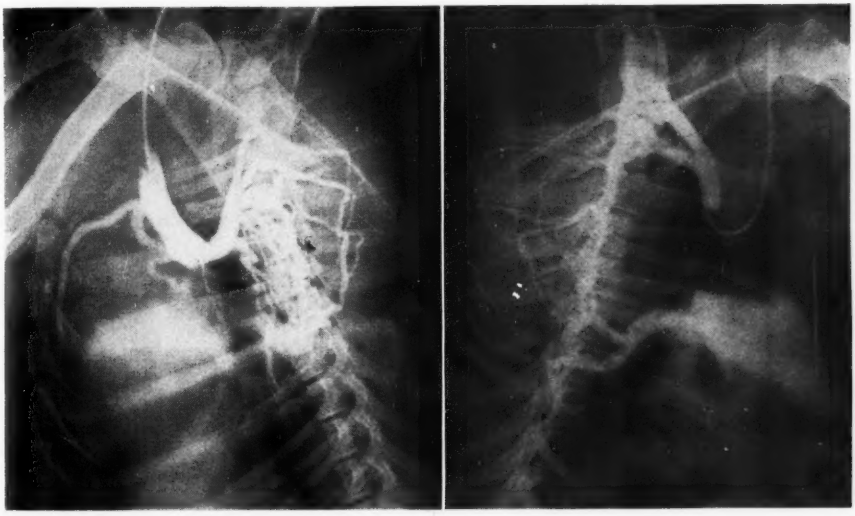
Temporary superior vena cava occlusion was produced in six dogs. Pressure recordings were obtained in five animals. The first animal had the superior vena cava obstructed for three hours and fifteen minutes, while the other five dogs had caval obstructions for two hours each. An arbitrary occlusion period of two hours was thought to represent a maximum period which might be required to effect an anastomosis in the clinical case. Pressure recordings were obtained by inserting polythene catheters (1.2 mm. in diameter) into the proximal portion of the superior vena cava, into the right auricle through the wall of the auricular appendage, and into the aorta either through the right subclavian or the femoral artery.

Control recordings were obtained prior to clamping the vena cava. Observations were recorded at the time the superior vena cava was obstructed by

clamps, at fifteen-minute intervals for two hours, at the moment of release of the caval obstruction, and for a short period thereafter.

At the completion of the experiment, the chest was closed in layers and the free air in the pleural cavity was aspirated as the lungs were re-expanded by positive endotracheal pressure. Procaine penicillin (300,000 units) was given intramuscularly to each animal after the operation. Postoperatively the animals were kept in cages in heated quarters and observed for survival time, cyanosis, and evidence of cerebral damage.

One animal had a venogram done thirty-five days after division and closure of the superior vena cava proximal to the right auricle. Fig. 1, *A* illustrates the venogram from this animal made by injection of 20 c.c. of 70 per cent Diodrast through the polythene catheter inserted into the left external jugular vein. A venogram immediately after superior vena caval obstruction was obtained in another animal (Fig. 1, *B*) revealing a readily available collateral venous return from the head and forequarters of the dog.



A. *B.*

Fig. 1.—*A*, Venogram obtained thirty-five days after acute permanent obstruction of the superior vena cava between the azygos vein and the right auricle in the dog. *B*, Venogram obtained within fifteen minutes after acute permanent obstruction of the superior vena cava between the azygos vein and the right auricle in the dog.

Experimental Results.—Six dogs had permanent acute obstruction of the superior vena cava with fatal results in 60 per cent of the animals within six to eighteen hours after operation. One of the fatal cases was complicated seriously by technical difficulties during dissection of the superior vena cava and death was probably more directly related to the excessive loss of blood. The two survivors in this group remain alive with moderate cyanosis of the mucous membrane and tongue 104 and 15 days, respectively, after caval obstruction.

As an initial exercise, temporary occlusion was effected for three hours and fifteen minutes in the first dog without recording pressure changes. Following the abrupt release of the clamp there was rapid distention of the auricle and the ventricles went into fibrillation from which they could not be extricated with the available electric defibrillator and death ensued.

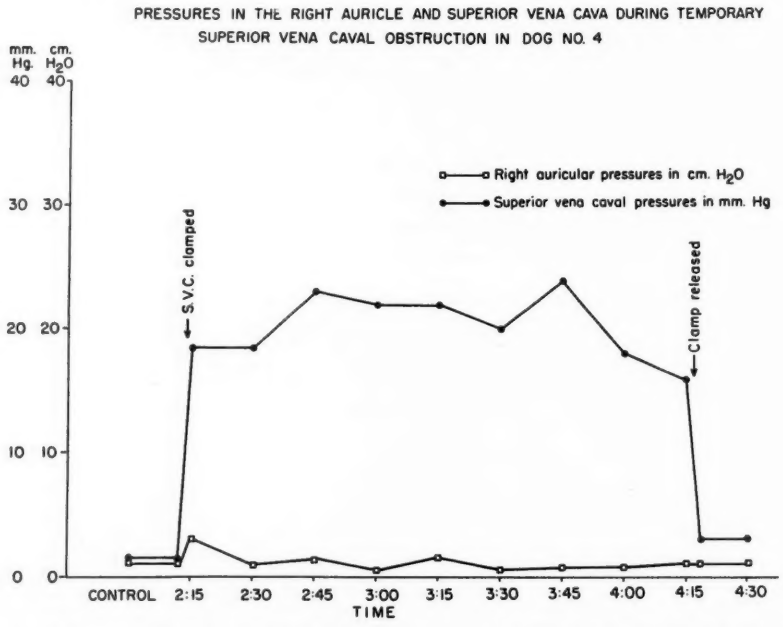


Fig. 2.—A graph showing the auricular pressures (in centimeter of water) and the superior vena caval pressures (in millimeter of mercury) in the control period, during caval occlusion, and after release of the occlusive clamp in the dog.

Temporary obstruction for two hours was similarly carried out in five additional dogs. In all of these animals there was a prompt elevation of pressure in the superior vena cava proximal to the point of obstruction and relatively little change in the right auricular pressure. Fig. 2 graphically illustrates the pressure alterations observed in Dog 4 after obstruction of the superior vena cava and immediately after its release. The electrocardiographic records did not reveal any significant arrythmia associated with superior caval obstruction or following its removal. This was contrary to our expectations in view of the reports by Carlson (1934) and the results obtained by us in the first animal in this series which died from ventricular fibrillation. Superior vena caval obstruction also did not produce significant alterations in the arterial blood pressure, an observation corroborating the findings of Andreasen and Watson (1952). Two dogs in this series died eighteen and eight hours, respectively, after operation from pulmonary atelectasis while the remaining two dogs were sacrificed while in good health one and six weeks, respectively, after temporary

superior caval obstruction. All the animals in this group had the site of occlusion and proximal superior vena cava examined for thrombus formation but none was found.

Fig. 3 is a graphic representation of the superior vena caval obstruction in five dogs. Dogs 3 and 4 were sacrificed while in good health. Dogs 2 and 5 expired with pulmonary complications in the early postoperative period.

In all of these animals cyanosis of the mucous membranes and tongue was very marked during the period of superior caval obstruction. The three dogs which survived one week or more did not show a sustained intense cyanosis. The two survivors from among the dogs with acute permanent caval obstruction showed a decrease of the cyanosis several days after operation. The two dogs which died with pulmonary complications may well have had cerebral damage secondary to temporary venal obstruction. There was evidence of cerebral damage in four dogs whose venae cavae were permanently occluded. Three died without gaining consciousness. The fourth regained consciousness but displayed a flaccid paralysis of all extremities.

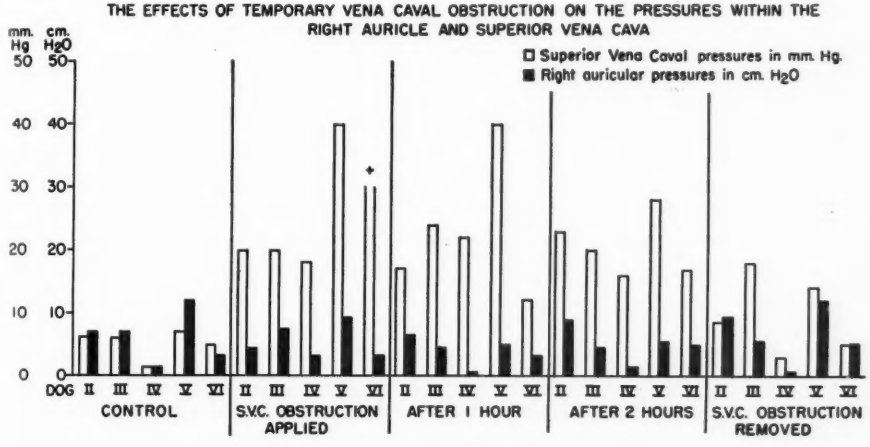


Fig. 3.—Graphic illustration of the right auricular and superior vena caval pressures with relationship to temporary acute obstruction of the superior vena cava in five dogs.

DISCUSSION

Occlusion of the superior vena cava at the cava-auricular juncture produces a rapid rise in the intracaval pressure to plateau levels two to three and one-half times greater than the occlusion level. Intense cyanosis occurs in the head, neck, and upper extremities as a result of the slowed venous return. That the maintenance of the high plateau pressure may, in the main, be a result of the immediate availability of the azygos system as an alternate route of return flow of the venous blood through the inferior vena cava is illustrated by the venogram (Fig. 1, *B*) taken fifteen minutes following acute permanent pre-

atrial superior vena caval occlusion. By comparison a similar venogram (Fig. 1, *A*) shows only slight further dilatation of these venous collaterals thirty-five days following the permanent caval occlusion. Alterations in the peripheral vascular bed that may further reduce the volumes of blood returning from the upper extremities can only be hypothecated. At any rate it would seem that the immediate availability of the azygos system to provide for collateral return of venous blood is an important factor in both limiting the height of the pressure rise proximal to the occlusion and possibly in permitting sufficient blood flow through the cerebral circulation to maintain cell viability for a considerable time in spite of intense cyanosis.

Similar experiments with the azygos vein tied and with the azygos intact but occluding the superior vena cava above it would be interesting. Andreasen and Watson (1952) clearly indicate the importance of the azygos vein when complete temporary obstruction of venous return to the heart is produced. We recognize certain fallacies in the comparison of the venous circulation in the extremities of the experimental animal to the human being, but feel that the caval and azygos systems, devoid of valves in both, should be similar.

The drop in intra-auricular pressure is much less dramatic than might at first be expected. Since in the dog 60 per cent of the venous return to the heart is through the inferior vena cava, the volume of blood available for auricular filling is still great even after superior caval occlusion. To this is added the sizable contribution of the azygos collateral blood flow. The resultant diminution in venous return is reflected in reduced cardiac output which is not sufficiently reduced markedly to lower the intra-aortic pressure. Temporary occlusion for two hours, though a serious physiologic shock, is tolerated by the dog surprisingly well. These observations bear out the results of Gerbode (1950), who found that occlusion of the superior vena cava was tolerated for twenty-five minutes in making experimental anastomoses between the superior vena cava and the left auricular appendage in dogs. The findings in our experiments would seem to bear out the impression from the single clinical case that patients might tolerate occlusion of the superior vena cava long enough to permit reconstructive anastomosis or even replacement by graft. The increasing frequency of major thoracic procedures with dissection of vessels about the pulmonary hilum within the pericardium, as described by Allison (1946), or of so-called "radical pneumonectomy," described by Cahan and his associates (1951), may increase the frequency of caval injury and the necessity for its reconstruction.

Permanent occlusion of the superior vena cava at this level presents a much more serious physiologic shock to the animal. It is, however, not universally fatal; two of the six animals survived. Those that died exhibited evidences of cerebral damage consequent to the selective cyanosis produced by the procedure.

Bailey (1952) has recently described the development of ventricular fibrillation following the release of simultaneously occluded superior and inferior

venae cavae. We had observed a similar occurrence in the first experiment following occlusion of the superior vena cava for over three hours and felt that ventricular fibrillation might be produced by this means fairly readily. In this our expectations were unfulfilled since no subsequent disturbances in cardiac mechanism occurred in any of the experimental animals following caval occlusion for two hours. It may require longer caval occlusion to produce ventricular fibrillation with any degree of certainty.

SUMMARY

A clinical case in which the superior vena cava was divided and re-implanted into the right auricle prompted experiments with occlusion of the cava to evaluate the resultant physiologic changes. Temporary occlusion at the cava-auricular junction of two hours' duration was tolerated by the experimental animal. The superior caval pressure rose rapidly to maintain a high plateau level two to three and one-half times greater than the preocclusion level. The azygos system provided an immediate route for collateral shunt around the occlusion. Pressures within the right auricle and the aorta fell only slightly. Permanent occlusion at this level, with an intact azygos system, was tolerated by two of the six dogs.

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INCLUSION DISEASE OF INFANCY

Case Report

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CYTOMEGALIC inclusion disease (also referred to as generalized salivary gland virus infection) is a systemic disease, characterized by the presence of intranuclear and intracytoplasmic inclusion bodies in gigantically enlarged cells of many organs. While predominantly an affection of infants and newborns, cases in older children and adults have been reported. Inclusion-bearing cells of similar morphology have been found in the ducts of salivary glands of 12 per cent of infants in a consecutive autopsy series,[1] probably representing latent infection or a commensal type of relationship. Although the so-called "salivary gland virus" is more or less universally accepted as the etiological agent, no report of its recovery or cultivation from clinical material has appeared to the present time.

A recent comprehensive review of the literature by Smith and Vellios[2] yielded sixty-nine cases, to which were added twenty new ones. All of these were discovered at autopsy. Many were associated with other diseases or debilitating conditions, which latter, rather than inclusion disease itself, seemed to be the proximate causes of death. A few occurred as isolated infections without recognized accompanying disease.

Until very recently no satisfactory diagnostic measures have been available, and for this reason figures as to incidence and mortality must be considered open to question. Fetterman[3] has recently described finding typical inclusion-containing cells in the urine sediment of an infant who subsequently died of inclusion disease; the disease was demonstrated at autopsy. While the application of this discovery may well lead to more frequent recognition, there has been to this time no description of a case in which the diagnosis has been established during the patient's life, and in which there has been clinical recovery. The present case is, therefore, unique in this respect and also in the concomitance of cystic lung disease, for which the preceding inclusion disease may reasonably be held responsible.

CASE REPORT

The patient, D. B., a white boy, was the first born of twins, delivered on Oct. 26, 1951. He weighed 2,300 grams, cried spontaneously, and exhibited normal vigor at birth. About the sixth day of life, his appetite began to wane. He was moderately jaundiced; the spleen extended 3 cm. below the costal margin and the liver was barely palpable. No other positive physical signs were found.

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On the following day, cyanosis appeared during bouts of crying, and the cry was weak and high pitched. The rectal temperature was elevated to 100° F. for a few days, then fell to normal. Cyanosis and dyspnea became progressively worse. About a week after the onset of symptoms, a chest x-ray film showed "a marked opacity over the upper half of the right lung, which represents either consolidation or atelectasis or both." (Fig. 1.) The first chest signs made their appearance on November 19, described by the observer as "decreased breath sounds over the right upper lobe area." By this time the baby had become ominously dyspneic and cyanotic, despite continuous oxygen administration. A loose cough, productive of small quantities of blood-tinged mucus, appeared; coarse râles became audible at the right base, and no normal alveolar breath sounds could be heard on the right side. The radiologic density had, by November 24, spread to involve the entire right lung field. The trachea and mediastinum were displaced to the right, and a small, sharply marginated area of radiolucency had appeared in the upper portion of the lower lobe.

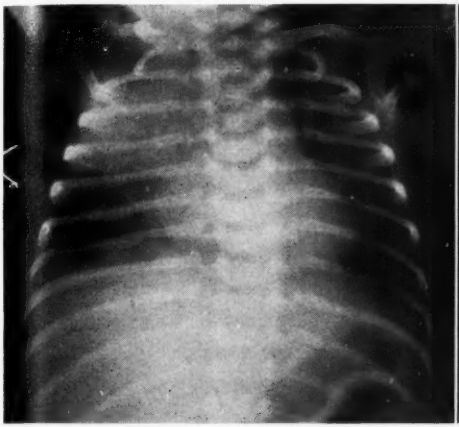

Fig. 1.

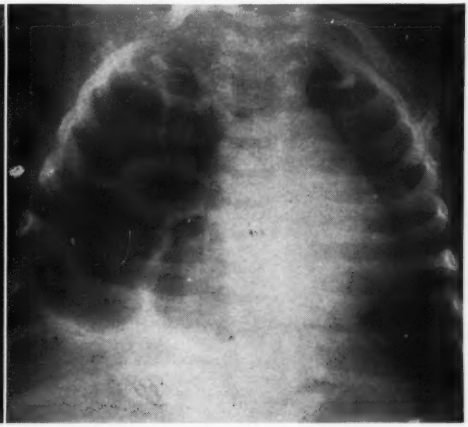

Fig. 2.

From this point onward, there was gradual improvement in the baby's general condition. The cough, dyspnea, and cyanosis subsided partially, but did not disappear. His appetite became slowly greater, and he began to gain weight in spite of obvious respiratory difficulty. Serial x-ray studies disclosed the development of four air-filled cysts at the site of the previously observed radiolucent area. These appeared to compress the adjacent lung tissue and displaced the mediastinum slightly to the left (Fig. 2).

The laboratory work done during the first two months of life revealed no specific pattern. The hemoglobin was 17.5 Gm./100 ml. at birth, gradually declined to 9.6 Gm./100 ml. The leukocyte count varied moderately, but was never significantly elevated, and the differential count was always within normal newborn limits. Only banal organisms were cultured from sputum and pharyngeal swabs. Blood cultures taken on several occasions during this period remained sterile for six weeks.

Because of progressive enlargement of the pulmonary cysts and because of continued dyspnea and cyanosis, surgery was undertaken. Thoracotomy was performed December 19, by J. W. S. Arising in the lower portion of the upper lobe and the apex of the lower lobe was a group of tensely distended, air-filled cysts which filled most of the right hemithorax. The remaining uninvolved portions of the lung lay collapsed and airless against the mediastinum. Resection of the affected segments was accomplished with little difficulty. The postoperative course was favorable; cyanosis disappeared and the need for oxygen dimin-

ished promptly. In later radiographs a small residual cystic area in the right cardiophrenic angle was visible. This has shown no enlargement since and does not seem to cause discomfort. The patient was discharged on March 5, 1952, in good condition. Since discharge, he has remained entirely well and is developing normally.

Microscopic Appearance.—The excised portions of lung showed moderate increase in interstitial tissue, with occasional areas of striking fibrosis in interalveolar septa and peribronchial tissues. A rather sparse infiltration of lymphocytes, plasma cells, and histiocytes was widespread in distribution. The cysts were lined by delicate layers of fibrin, with surrounding thin laminas of young connective tissue. In several of the sections, communication of cysts with small bronchi could be definitely established.

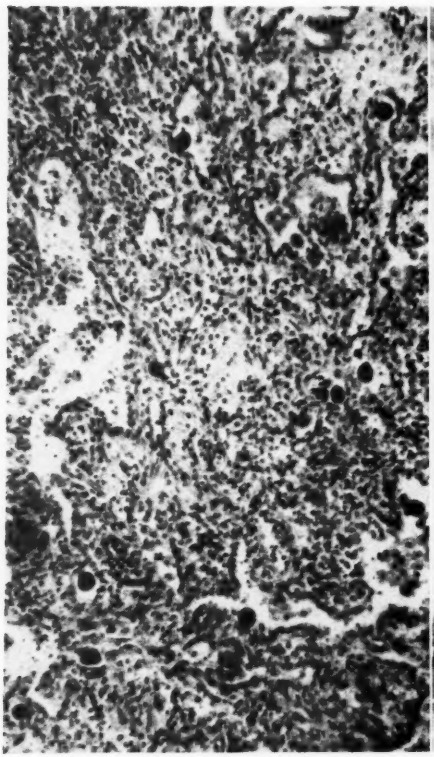

Fig. 3.

Fig. 4.

Scattered throughout all regions of the lung tissue were large cells, 17 to 24 microns in diameter, containing both intranuclear and intracytoplasmic inclusion bodies (Fig. 3). These cells lay largely within the lumina of terminal bronchioles and were apparently derived from bronchiolar lining cells. The intracytoplasmic inclusions consisted of crescent-shaped groups of faintly basophilic granules lying at the periphery of the gigantic cells. Within each nucleus was a large spherical, homogeneous, reddish-purple inclusion, 7 to 8 microns in diameter, almost filling the entire structure. A narrow clear space lay between the border of the inclusion and the peripherally clumped nuclear chromatin in about one-half of the cells. In the remainder the clear space was obliterated, the margins of the inclusions being indistinct

(Fig. 4). This phenomenon has been regarded as representing degeneration of the virus and regression of disease, a concept which coincides with the clinical status of this case at the time the lung was resected.

In one tissue block was found, projecting into a small bronchus, a nodule, composed of moderately dense, richly vascular, fibrous stroma, imbedded in which were myriad giant inclusion-bearing cells. Bronchial mucosa covered the base of the polypoid structure, but was absent over its crest (Fig. 5). Practically all of the intranuclear inclusions in the nodule had indistinct margins as previously described. The size, location, and structure of the lesion implied that it had obstructed the bronchiole it occupied at least partially, and possibly had had a ball-valve effect.

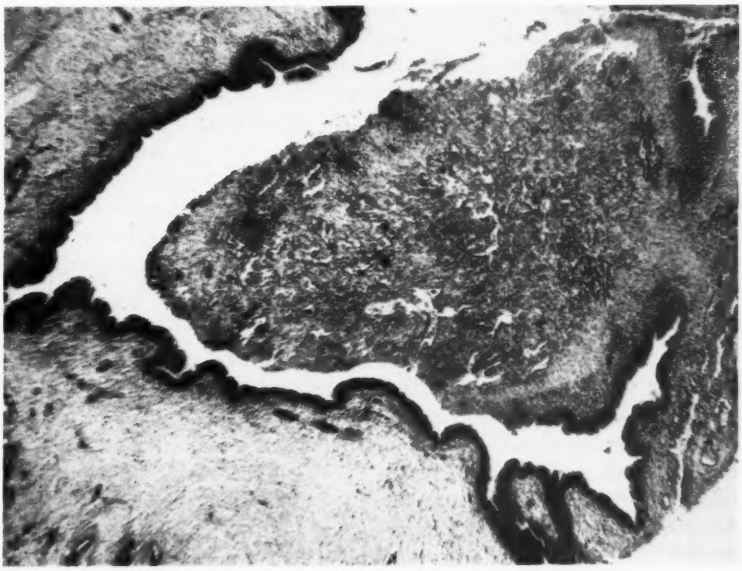

Fig. 5.

DISCUSSION

This case reveals certain facets of the nature of cytomegalic inclusion disease previously suspected, but not clinically demonstrated. Two points deserve special comment. The first of these is the simple fact that the patient recovered. Although no previous reported case has been proved prior to autopsy, this probably is more the result of lack of diagnostic measures and the tendency of the disease to be coexistent with serious conditions than an indictment of it as a universally fatal infection. Nonfatal cases almost certainly occur, perhaps with greater frequency than more serious forms. The use of cytologic studies as suggested by Fetterman may lead to more frequent diagnosis of milder instances of the disease and their segregation from that unsatisfactorily vague category, "virus infection."

The other noteworthy point in the present case is that there appeared structural changes with strong indication that this was the result of the pre-

ceding virus infection. Such relationship is also suggested in Cases 11 to 15 of Smith and Vellios, all of whom had fibrocystic disease of the pancreas associated with cytomegalic inclusion disease. Whether these entities, and other similar disorders of late childhood and adult life may frequently be the sequelae of salivary gland virus infection can only be proved or disproved by the accumulation and study of an adequate series of healed cases of inclusion disease.

It is believed that, in the child described here, the illness began as a primary infection with the salivary gland virus, acquired either in utero, or more likely in the immediate postnatal period. Prematurity may well have been a predisposing factor. Bronchial and bronchiolar swelling, with or without the formation of intrabronchial polypoid granulomata, is credited as having been the direct cause of the pulmonary cysts which later developed. Changes in interalveolar supportive tissue were probably contributory. Regression of the acute phase of the disease, either spontaneously or under the influence of antibiotics, began about four weeks after the onset of symptoms. At the time of lobectomy, the disease was in its terminal phase. Had a greater period of time elapsed before operation, it is entirely possible that all evidences of the foregoing inclusion disease would have vanished, leaving only a cystic lung of cryptic etiology.

SUMMARY

A case is described in which a newborn infant developed pulmonary cysts following an episode of pneumonitis shortly after birth. The excised portions of lung showed the histologic picture of cytomegalic inclusion disease. The patient recovered without incident. This is believed to be the first reported case of cytomegalic inclusion disease in which recovery has occurred. It is also significant that in this case structural lung changes were apparently the result of salivary gland virus infection.

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BRONCHIOGENIC CARCINOMA

A Statistical Review of Two Hundred and Thirty-Four Autopsies

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OBJECTIVE

A GROUP of 234 consecutive autopies on cases of bronchiogenic carcinoma are reviewed in this paper to determine the different clinical types and the most common sites of metastases. The literature is also reviewed.

DEFINITION

It is the widely accepted conviction that all primary pulmonary malignancies of epithelial origin arise from the bronchi or their accessory glands. Such malignancies are therefore termed bronchiogenic carcinoma.

HISTORY

Johannes Agricola, in the sixteenth century, first described the symptoms of bronchiogenic carcinoma. The pathology of pulmonary tumors was first described in 1766 by Morgagni who also gave a full autopsy report in which it is possible to recognize the present-day descriptions of lung carcinoma.

Boyle, in 1810, reported three cases of primary cancer of the lung and noted diffuse and nodular types.

Laennac and his co-workers in 1819 described "encephaloid" of the lung, but they were not able to diagnose it ante mortem. Stokes in London in 1837 stated that frequent and severe attacks of bronchitis, resisting the usual methods of treatment, is suspicious of lung malignancy. He also gave an account of the physical signs, the qualities of the sputum, and the differential diagnosis between these signs and those of aneurysm. In 1843, Walsche termed this condition "cancer." In the same year, Kohler, of Germany, wrote a monograph on the subject. In 1842, Stokes made the first diagnosis by physical examination and gave a complete description of its clinical manifestations. Virchow, in 1863, first described the pathologic process of pulmonary tumors, including cancer of the lung. It was his opinion that primary cancer of the lung was rare.

Passler in 1896 found 57 collected cases while Adler in 1911, in a study of primary bronchiogenic carcinoma, found 360 cases reported in the literature. The latter concluded the failure to recognize lung cancer had given rise to the erroneous impression that it was a rare malignancy.

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HISTOPATHOLOGY

The exact origin or cell derivation of each particular type of bronchiogenic carcinoma is not entirely settled, but suffice it to say we believe that the epidermoid and undifferentiated carcinomas arise from the basal cell layer and the adenocarcinomas arise from the bronchial glands.

We attempted to simplify our classification by using three main categories, each with its individual subdivisions. We included in the epidermoid group the squamous-cell carcinoma with and without pearl formation and the undifferentiated squamous-cell type. These are made up of large eosinophilic staining cells, appearing polyhedral and platelike with a relatively small nucleus rich in chromatin. The adenocarcinomas included mucus- and nonmucus-producing types. The undifferentiated group, which we feel is the most malignant, includes the so-called "oat-cell," round-cell, and small-cell carcinomas. Microscopically, it is a small ovoid cell with a large nucleus, rich in chromatin, and appearing like a lymphocyte in fixed hematoxylin and eosin material. These cells closely resemble lymphosarcoma cells. We feel that any further differentiation is a matter of personal impression and interpretation. These various groups occasionally occur in combination with one cell type predominating.

We found no cases that would satisfactorily meet any or all of the criteria established for the diagnosis of the so-called alveolar-cell carcinoma. We were able to find a primary in the main stem bronchus with dropped metastases in the alveoli in several cases which gave the impression of a so-called alveolar-cell carcinoma. In more recent literature the so-called alveolar-cell carcinoma has been termed adenomatosis and is felt not to be in the carcinoma realm.

SITES OF METASTASES

The problem of metastases from malignant pulmonary tumors is of major importance since the employment of lobectomy and pneumonectomy in its treatment. The routes of spread are not many, but these must be considered: (1) lymphogenous spread; (2) hematogenous spread, including the vertebral veins; (3) direct extension, (4) implantation, following such procedures as aspiration, aspiration biopsy, and operation, and (5) bronchiogenic spread.

It is believed that the frequent occurrence of metastasis in carcinoma of the lung is due to the fact that the lungs have such a rich vascular and lymphatic bed. There is a greater tendency for bronchiogenic carcinoma to be disseminated throughout the body by the invasion of blood vessels than carcinomas arising elsewhere.

Carcinomas arising elsewhere liberate emboli into systemic veins which are caught or filtered out in the lung, while those liberated in the portal venous system find their way into the liver. Those liberated from the lungs into the pulmonary veins, or into the left chamber of the heart, will be caught in any peripheral organ to which they are carried.

The usual tendency is to invade regional lymphatics, thus the parabronchial and peritracheal nodes first, then the mediastinal lymph nodes. Early metasta-

TABLE I. HISTOLOGIC TYPES

	HINES 1942-1947		KOLETSKY[1] 1936		ARKIN AND WAGNER[2] 1936		TENZEL[3] 1936	
Total No. of cases	234		125		74		192	
Epidermoid	145	61.96%	58	46.4%	21	28%	105	54.58%
Adenocarcinoma	21	8.97%	33	26.4%	18	24%	50	25.04%
Undifferentiated	68	29.06%	34	27.2%	31	43%	37	19.27%

sis is the rule in primary bronchiogenic carcinoma. Signs of metastases may be the presenting symptom or symptoms of the patient and may appear early.

Our series showed 61.69 per cent epidermoid carcinoma; 8.97 per cent adenocarcinoma; and 29.06 per cent undifferentiated. This demonstrated that 6 out of 10 were epidermoid carcinoma, 3 out of 10 were undifferentiated and 1 out of 10 was an adenocarcinoma.

TABLE II. SITE OF ORIGIN

Right side	HINES 1942-1947		KOLETSKY[1] 1936		Left side	HINES 1942-1947		KOLETSKY[1] 1936	
Total No.	138		81			96		44	
Epidermoid	86	62.31%	37	45.68%		58	61.41%	21	47.73%
Adenocarcinoma	12	8.69%	22	27.16%		10	10.41%	11	25.00%
Undifferentiated	40	28.98%	22	27.16%		28	28.11%	12	27.27%

We found the right side more frequently involved than the left (58.9 per cent originated on the right side, which was a little over one-half.) This may, in all probability, be due to the fact that the right main stem bronchus is an almost direct continuation of the trachea.

The epidermoid variety originated most frequently on the right side; the undifferentiated group was practically equally divided with the right side still somewhat more predominant, while the adenocarcinoma group were equally divided.

TABLE III. COMPARATIVE SERIES OF INDIVIDUAL SIDES

	HINES	OCHSNER AND DE BAKEY[4]	FISCHER[4]	ARKIN AND WAGNER[2]	KOLETSKY[2]
Total No. of cases	234	4732 (Collected)	3735	135	125
Right side %	58.97%	58.3%	53%	60%	64.8%
Left side %	41.03%	41.6%	45%	40%	35.2%
Bilateral %			2%		

Ochsner and DeBakey, in 1941, reported a series of 4,732 in which 58.3 per cent were on the right side and 41.6 per cent were on the left. Fischer, in 1940, reported 3,735 collected cases in which there were 53 per cent on the right side; 45 per cent on the left side; and 2 per cent were bilateral. Arkin and Wagner, in 1936, reviewed 135 cases of which 60 per cent originated on the right side, and 40 per cent on the left side. Koletsky, in 1936, had 125 cases of which 64.8 per cent were on the right side and 35.2 per cent originated on the left side. Our series showed 58.97 per cent on the right side with 41.03 per cent on the left side.

TABLE IV. LYMPH NODE METASTASES

			EPIDERMOID			ADENOCARCINOMA			UNDIFFERENTIATED		
	NO.	% OF TOTAL	NO.	% OF MET.	% OF EPID.	NO.	% OF MET.	% OF ADENO.	NO.	% OF MET.	% OF UNDIFF.
Peribronchial	126	53.85	70	55.56	48.28	12	9.52	57.14	44	34.91	64.71
Mediastinal (including peritracheal)	98	41.88	54	55.10	37.24	10	10.20	47.62	34	34.69	50.00
Abdominal (including retroperitoneal)	42	17.95	23	54.76	15.86	3	7.14	14.29	16	38.09	22.06
Cervical	18	7.69	13	72.22	8.97	2	11.11	9.52	3	16.67	4.41

Lymph nodes showed the greatest involvement in our series. There were 126 of the 234 cases which showed peribronchial lymph node metastases. This was 53.85 per cent of the total number of cases. Of this 126, 70 (55.56 per cent) of the total were epidermoid carcinoma, while 48.28 per cent of the total number of epidermoid cases demonstrated peribronchial involvement. We also found that 12 (9.52 per cent) cases were adenocarcinoma, constituting 57-14 per cent of the total number of the adenocarcinomata. The undifferentiated category was made up of 44 (34.91 per cent) cases showing peribronchial metastases but this constituted 64.71 per cent of this cell type.

Mediastinal including peritracheal glands were involved in 98 cases, 41.88 per cent of the total number. Of these 54 (55.10 per cent) were epidermoid, this being 37.24 per cent of this particular category; 10 (10.20 per cent) were adenocarcinomas, constituting 47.62 per cent of the total number of adenocarcinomas. Thirty-four (34.69 per cent) of the undifferentiated group showed involvement, this making up 50 per cent of the total number of the undifferentiated cell type.

Forty-two cases (17.95 per cent) in our series demonstrated abdominal and retroperitoneal lymph node involvement.

Metastases to cervical lymph nodes occurred in 18 cases. No involvement of axillary lymph nodes was demonstrated.

TABLE V. METASTASES TO THE LIVER

	TOTAL NO.	% OF TOTAL		SAMPSON[5] 1935		TENZEL[3]		KOLETSKY[1]
HINES 1942-1947	90	38.46	% OF CELL TYPE	35	% OF CELL TYPE	74	% OF CELL TYPE	% OF CELL TYPE
Epidermoid	48	53.33	33.10	4	11.42	29	28	18
Adenocarcinoma	9	10.00	42.86	20	57.14	27	54	50
Undifferentiated	33	36.67	48.53	11	31.43	18	49	66

Hepatic metastases were found as frequently as some of the other more commonly expected sites. We had a total of 90 (38.46 per cent) in which liver metastases were present; 48 cases (53.33 per cent of the total) or 33.10 per cent of the epidermoid class. Nine cases of adenocarcinomata (10 per cent of the total) or 42.86 per cent of this cell type, and the undifferentiated group comprised 33 cases (33.67 per cent of the total) while this was 48.53 per cent of the cell type.

Sampson, in 1935, reported 35 cases with metastases to the liver. He broke this number down showing 4 cases of epidermoid which was 11.42 per cent; while 20 cases or 57.14 per cent were adenocarcinomas, and the undifferentiated group constituted 11 cases or 31.43 per cent.

Tenzel reported 74 cases in which he reported 29 epidermoid, 27 adenocarcinoma, and 18 as undifferentiated carcinomas, 49 per cent of this cell type.

Koletsky in 1936 reported 125 cases, 18 per cent epidermoids, 50 per cent adenocarcinoma, and 66 per cent in the undifferentiated group showed hepatic metastases.

TABLE VI. ADRENAL METASTASES

	HINES			TENZEL[3]		SAMPSON[5]		KOLETSKY[1]
	TOTAL NO. 89	% OF TOTAL 38.03	% OF CELL TYPE		% OF CELL TYPE		% OF TOTAL	% OF CELL TYPE
Epidermoid	50	56.18	34.48	21	20	3	14.29	54.0
Adenocarcinoma	11	12.36	52.38	22	44	16	76.19	12.0
Undifferentiated	28	31.45	41.18	16	45	2	9.52	64.0

GAINES	OCHSNER AND DE BAKEY[4]	MILLER AND JONES[5]	ARKIN AND WAGNER[2]	MATTICK AND BURKE[6]	GRAHAM[7]	HINES
14%	17.6%	9.7%	43%	8%	33.3%	38.03%

Almost 2 out of every 5 bronchiogenic carcinomata have adrenal involvement. This may be the chief reason that some of the people whom we see in an advanced state appear so debilitated. Of this group 1 out of 3 is squamous cell; 1 out of 8 is adenocarcinoma; and 1 out of 3 undifferentiated.

TABLE VII. KIDNEY METASTASES

	HINES			SAMPSON[5]		TENZEL[3]		KOLETSKY[1]
	TOTAL NO.	% OF TOTAL	% OF CELL TYPE	TOTAL NO.	% OF TOTAL	TOTAL NO.	% OF CELL TYPE	% OF CELL TYPE
	66	28.21		23		35		
Epidermoid	38	57.58	26.21	2	9.69	14	13	15
Adenocarcinoma	9	13.64	42.85	17	73.91	14	28	36
Undifferentiated	19	28.78	26.47	4	17.39	7	19	20

	HINES	OCHSNER AND DE BAKEY[4]	MULLIGAN AND HARPER[8]	ARKIN AND WAGNER[2]	MILLER AND JONES[4]
Total no. of cases	234	2579	44	74	808
No. of cases with metastases	66	412	11	29	122
% of total	28.21	16	25	40	15.9

We found 28 per cent involvement of the kidney suggesting that almost 1 out of every 3 has renal metastases. The epidermoid carcinoma metastasized to the kidney in 1 out of 4 cases; the adenocarcinomas in about 1 out of 7; and the undifferentiated group in about the same proportions as the epidermoids.

Sampson in 1935 reported 23 cases of which 2 or 9.69 per cent were epidermoid carcinoma; 17 or 73.91 per cent were adenocarcinoma; and 4 cases of 17.39 per cent were of the undifferentiated group.

TABLE VIII. BONE METASTASES

	HINES			SAMPSON[5]		KOLETSKY[1]
	TOTAL NO.	% OF METAS.	% OF CELL TYPE	TOTAL NO.	% OF METAS.	% OF CELL TYPE
	41	17.52		30		
Epidermoid	26	63.41	17.93	8	26.67	10
Adenocarcinoma	6	14.63	28.57	17	56.67	32
Undifferentiated	9	21.95	13.24	5	16.66	23

	HINES	OCHSNER AND DE BAKEY[4]	ARKIN AND WAGNER[2]	MILLER AND JONES[4]
Total no. of cases	234	2,579	74	808
No. of cases showing metastases	41	527	21	88
% of metastases	17.52	20.4	28	10.1

Tenzel in 1936 had 35 cases in which he reported renal involvement; 14 epidermoid carcinoma; 14 adenocarcinoma; and 7 in the undifferentiated group.

Koletsky in 1936 reported 15 per cent epidermoid carcinoma; 36 per cent adenocarcinoma and the undifferentiated group, constituting 20 per cent.

Bony metastases are not being reported as individual osseous sites, but are grouped together in a single total.

TABLE IX. LUNG METASTASES

	HINES			TENZEL[3]		KOLETSKY[1]
	TOTAL NO.	% OF METAS.	% OF CELL TYPE	TOTAL NO.	% OF CELL TYPE	% OF CELL TYPE
	34	14.57		52		
Epidermoid	21	61.47	14.48	20	19	12
Adenocarcinoma	5	14.71	23.81	23	46	55
Undifferentiated	8	23.52	11.77	9	24	11

	HINES	MILLER AND JONES[4]	ARKIN AND WAGNER[2]	SAMPSON[5]
Total no. of cases	234	808	74	40
Total no. showing metastases	34	173	24	10
Per cent of total	14.57	21	32	25

TABLE X. METASTASES TO HEART, PERICARDIUM, AND MAJOR VESSELS

	HINES			TENZEL[3]			SAMPSON[5]		KOLETSKY[1]
	TOTAL NO.	% OF TOTAL	% OF CELL TYPE	TOTAL NO.	% OF TOTAL	% OF CELL TYPE	TOTAL NO.	% OF TOTAL	% OF CELL TYPE
	26	11.11		14	7.29		25		
Epidermoid	18	69.23	12.41	8	57.14	8	11	44	2.5
Adenocarcinoma	0	0.00	0.00	4	28.57	8	6	24	23.0
Undifferentiated	8	30.77	11.76	2	14.28	5	8	32	5.7

	HINES	OCHSNER AND DE BAKEY[4]	MULLIGAN AND HARPER[8]	ARKIN AND WAGNER[2]	MATTICK AND BURKE[6]
Total no. of cases	234	2,579	44	74	808
No. of cases with metastases	26	268	5	10	22
Per cent of total	11.11	10.4	11.36	14	2.7

There is no predilection for any particular bony structure. We found almost every bone in the body involved except the hands and feet. There was 1 out of every 6 cases which showed bony metastases with the epidermoid group predominating; the undifferentiated group was second; and the adenocarcinoma last.

The lung was involved by metastases in 1 out of every 7 cases with the epidermoid group still predominant. The undifferentiated group was second in number, while the adenocarcinoma still made up the smallest group.

Mediastinal structures were involved in 11 per cent of our cases, the epidermoid carcinoma predominated, with the undifferentiated group being second. We found no cases of adenocarcinoma.

TABLE XI. METASTASES TO THE PLEURA

	HINES			TENZEL[3]		SAMPSON[5]	
	TOTAL NO.	% OF TOTAL	% OF CELL TYPE	TOTAL NO.	% OF CELL TYPE	TOTAL NO.	% OF TOTAL
	26	11.11		43		45	
Epidermoid	15	57.64	10.34	18	17	15	33.33
Adenocarcinoma	3	11.54	14.29	16	32	18	40.00
Undifferentiated	8	30.77	11.76	9	24	12	26.67

	HINES	MILLER AND JONES[4]	MULLIGAN AND HARPER[8]	ARKIN AND WAGNER[2]
Total no. of cases	234	808	44	74
No. of cases with metastases	26	285	14	33
% of total with metastases	11.11	35.2	31.82	45

The pleura was involved in 11 per cent, with the epidermoid variety predominant.

TABLE XII. BRAIN METASTASES

	HINES			TENZEL[3]		KOLETSKY[2]		SAMPSON[5]	
	TOTAL NO.	% OF TOTAL	% OF CELL TYPE	TOTAL NO.	% OF CELL TYPE	TOTAL NO.	% OF CELL TYPE	TOTAL NO.	% OF TOTAL
					(% total)				
	19	8.11		23	11.98	10		15	
Epidermoid	11	57.89	7.59	6	6	1	5.3	2	13.33
Adenocarcinoma	3	17.59	14.29	14	28	5	36.0	12	80.00
Undifferentiated	5	26.31	7.50	3	8	4	33.0	1	6.67

	HINES	OCHSNER AND DE BAKEY[4]	MILLER AND JONES[4]	ARKIN AND WAGNER[2]	FRIED AND BUCKLEY
Total no. of cases	234	2,579	808	74	37
No. of cases with metastases	19	377	77	18	15
Per cent of total	8.1	14.6	9.5	24	40.54

Experimental work done at our hospital has shown that the vertebral veins are a more common avenue of metastasis to the brain and central nervous system than was believed.

Craig, Waltman, and Kernohan in 1938 stated that a metastatic tumor to the central nervous system may be the first indication of a malignancy else-

where in the body. Central nervous system metastasis may come from a tumor in almost any organ of the body, but the lung is the most frequent site of such a malignancy.

The first clinical manifestations of bronchiogenic carcinoma may be due to intracranial metastases, simulating a symptom complex produced by a rapidly growing and highly invasive primary tumor of the brain. King and Ford after reviewing 100 cases at Johns Hopkins concluded that carcinoma of the lung metastasizes to the brain more frequently than any other type of malignancy. This is due to the malignant cells being carried by the blood stream to the brain without passing through the capillary bed of the lungs. They reported that of 448 cases 52, or 12 per cent, showed symptoms referable to the central nervous system rather than the bronchus. There were 19 cases or 4 per cent in which the symptoms referable to the central nervous system were the presenting ones. The type of carcinoma was not a factor in the incidence of metastasis to the central nervous system.

There were 19 cases (8.11 per cent) of demonstrable brain metastases in our series. This indicated 1 out of every 12 had such involvement. In some clinics as high as 1 out of 5 has shown brain involvement. Here again the epidermoid class predominated, while the undifferentiated group was second, and the adenocarcinoma third.

TABLE XIII. MISCELLANEOUS

			EPIDERMOID			ADENOCARCINOMA			UNDIFFERENTIATED		
	TOTAL NO.	% OF TOTAL	TOTAL NO.	% OF TOTAL	% OF CELL TYPE	TOTAL NO.	% OF TOTAL	% OF CELL TYPE	TOTAL NO.	% OF TOTAL	% OF CELL TYPE
Pancreas	17	7.26	5	29.41	3.45	0	0.00	0.00	12	70.59	17.78
Spleen	14	5.55	7	50.00	4.82	1	7.14	4.76	6	42.86	8.82
Skin	8	3.41	6	75.00	4.14	0	0.00	0.00	2	25.00	2.84
Bowels (large and small)	5	2.13	2	40.00	1.38	0	0.00	0.00	3	60.00	4.41
Prostate	3	1.28	1	33.33	0.69	0	0.00	0.00	2	66.67	2.94
Diaphragm	2	0.85	1	50.00	0.69	0	0.00	0.00	1	50.00	1.47
Thyroid	2	0.85	1	50.00	0.69	0	0.00	0.00	1	50.00	1.47
Testicle	1	0.43	0	0.00	0.00	0	0.00	0.00	1	100	1.47
Gall bladder	1	0.43	1	100	0.69	0	0.00	0.00	0	0.00	0.00

This table includes other organs of the body in which metastases were found. We had 17 cases with involvement of the pancreas. This was 7.26 per cent of the total number of cases. There were 5 cases (3.45 per cent) of epidermoid carcinoma, 29.41 per cent of the total number which demonstrated metastases. There were no cases of adenocarcinoma. The undifferentiated group had 12 cases (17.78 per cent) of 70.59 per cent of the total number.

The spleen was involved in 14 cases; there being an almost equal number of epidermoid carcinoma and undifferentiated. There were 7 (4.82 per cent) cases of the former which constituted 50 per cent of the total number of cases. In the latter group there were 6 cases (8.82 per cent), 42.86 per cent of the total. In the adenocarcinoma group there was only 1 case (4.76 per cent) constituting 7.14 per cent of the total.

SUMMARY

TOTAL NO. CASES	HINES 234		OCHSNER AND DE BAKEY 4,732		ARKIN AND WAGNER 74		KOLETSKY 125		SAMPSON 40		TENZEL 192		MILLER AND JONES 808		MULLIGAN AND HARPER 44	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
Site of origin Rt.	138	58.97	2,749	58.3	45	60.00	81	64.8								
Lt.	96	41.03	1,383	41.6	29	40.00	44	35.2								
Liver	90	38.46	1,577	33.3			50	40.0	35	87.5	74	38.50	248	30.00	21	47.73
Adrenals	89	38.03	833	17.6	32	43.00	47	37.6	29	72.5	59	30.72	79	9.70	19	43.18
Kidney	66	28.21	412	16.0	29	40.00	27	27.6	23	57.5	35	18.23	122	15.90	11	25.00
Bone	41	17.52	526	20.4	21	28.00	25	20.0	11	27.5			88	10.10	15	34.10
Lung	34	14.57			24	32.00	29	23.2	10	25.0	52	27.08	173	21.00	9	20.45
Heart																
Pericardium	26	11.11	620	13.1	31	41.87	15	12.0	2	5.0	59	30.72	22	2.70	5	11.36
Major vessels																
Pleura	26	11.11			33	45.00					43	27.60	285	35.20	14	31.82
Brain	19	8.1	653	13.8	18	24.00	10	5.2	15	37.5	23	11.90	77	9.50	18	40.97
Miscellaneous	53	22.65			52	70.20	64	51.2	13	32.5	117	60.94	37	4.58	21	47.72

There were 8 cases or 3.41 per cent of the total number of cases in which metastases to the skin were found. It is stated that metastatic nodules in the skin should be considered from the bronchus until proved otherwise. Six (4.14 per cent) were of the epidermoid carcinoma group 75 per cent of the total number. There were no cases in which adenocarcinoma was found. Two (2.84 per cent) were in the undifferentiated classification constituting 25 per cent of the total.

Metastases to the bowel are infrequent, but do occur. We had 5 cases or 2.13 per cent to the large and small bowels. The small bowel, especially the duodenum, was the most frequent site of involvement. Two cases (1.38 per cent) or 40 per cent of the total were epidermoid carcinoma, while the undifferentiated class constituted 3 (4.41 per cent) cases or 60 per cent adenocarcinoma was conspicuous by its absence.

There were 3 cases of metastases to the prostate, 1.28 per cent of the entire number of cases. There was 1 (0.69 per cent) in which epidermoid was the predominating cell type; no cases in which adenocarcinoma metastasized; the undifferentiated carcinoma metastasized in 2 cases (2.84 per cent) or 66.67 per cent of the total.

The diaphragm was involved in only 2 cases. This was equally divided between the epidermoid and undifferentiated group. This constituted 0.69 per cent of the former cell type, and 1.47 per cent of the latter cell type.

The thyroid gland was involved in 2 cases or 0.85 per cent in which there was 1 case (0.69 per cent) of epidermoid carcinoma and 1 case (1.47 per cent) of undifferentiated carcinoma.

There was 1 case of metastases to the testicle which was 0.43 per cent of the total number of cases. This was a case of an undifferentiated carcinoma or 1.47 per cent of this cell type number.

The gall bladder was involved in only 1 case or 0.43 per cent of the total number of cases. This was in the epidermoid class or 0.69 per cent.

CONCLUSIONS

1. Metastases play an important role in the treatment of bronchiogenic carcinoma since the advent of surgery for this condition.

2. There is no organ or structure in the body which escapes metastases.

3. Metastases may occur in any of the established ways: (1) direct extension, (2) lymphogenous dissemination, (3) hematogenous dissemination (including vertebral veins), (4) direct implantation, and (5) bronchiogenic spread.

4. Carcinoma of the lung occurs with almost equal incidence on either side.

5. The simplest classification is epidermoid, adenocarcinoma, and undifferentiated. However, combinations of these do occur and would be difficult to fit into this arbitrary categorization.

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PULMONARY BLASTOMYCOSIS

The Influence of the Immunologic Findings on the Selection of Patients for Operation

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INFECTIONS of *Blastomyces dermatitidis* in man are serious; and, when disseminated, the survival rate is only 7 per cent.[2, 3] In some cases the only residual clinical manifestation of a generalized infection may be in the lung. Efforts in the past directed toward the surgical treatment of the pulmonary form of the infection have been almost uniformly poor.[1, 4, 5] In spite of this poor record it was thought that there were certain instances where careful immunologic evaluation of the patient might make the deliberate surgical excision of these localized pulmonary lesions feasible. In Duke Hospital two such cases were submitted to surgery. A report of this experience is recorded in this paper.

CASE REPORTS

Case 1.—C. W. Four months before admission to Duke Hospital this 36-year-old white man developed a severe respiratory infection that was associated with severe left anterior chest pain. Shortly thereafter he began to cough and spit up blood. Afternoon temperature elevation and profuse night sweats then developed. After this the patient's course was steadily downhill, in spite of treatment with penicillin, various sulfa compounds, and Aureomycin.

Past history and review of systems did not reveal anything of importance. The patient was a farmer.

Physical examination on admission showed a temperature of 38° C., pulse 100, blood pressure 106/62, and respirations 18. The chest examination showed signs of consolidation in the right upper lung field. The urinary tract and skeletal system were normal.

Laboratory studies revealed a hemoglobin of 10 Gm., white blood count of 14,000 with 80 per cent segmented forms, 3 per cent eosinophils, 6 per cent monocytes, 10 per cent small lymphocytes, and 1 per cent large lymphocytes. The urine showed a specific gravity of 1.010 with albumin, sugar, and microscopic sediment study negative. Blood chemistry showed total plasma proteins 7.3 Gm., albumin 2.9 Gm., and globulin 4.4 Gm. Examination of numerous sputum smears showed typical *Blastomyces dermatitidis* organisms. The complement fixation test was negative. Repeated skin tests with blastomycin were negative. Other skin tests with histoplasmin and coccidioidin were negative; but a 1:1000 old tuberculin skin test was positive.

X-ray examination of the chest showed a consolidated area in the left lung field which was confined mainly to the anterior and lingular segments of the left upper lobe (Fig. 1).

On bronchoscopic examination purulent secretions were noted coming from the left upper lobe.

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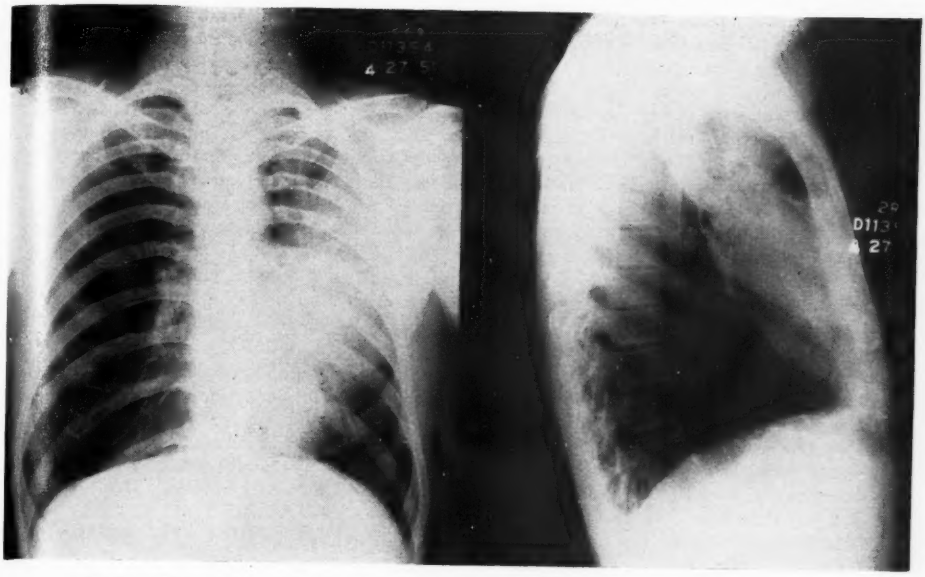

Fig. 1.—Films of patient C. W. with *Blastomyces dermatitidis* infection (Case 1).

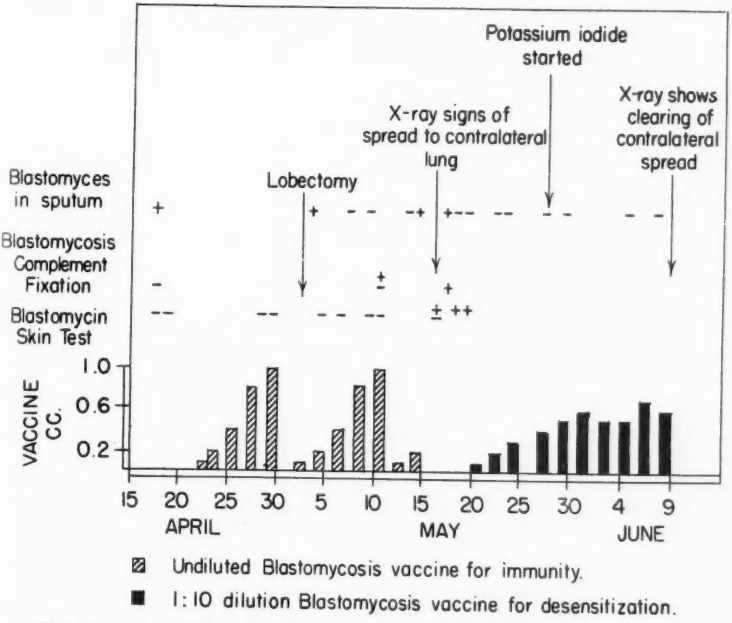

Chart 1.—The chart shows relationship of skin sensitivity, complement fixation, and presence of organisms in sputum to vaccine therapy, operation, and iodide administration in Case 1 (C. W.).

Hospital Course.—As seen in Chart 1 immunization with blastomyces vaccine was begun one week after admission because of the negative skin test and negative complement fixation. The schedule of vaccine administration was 0.1 c.c. increasing by 0.1 c.c. every other day until a daily dose of 1 c.c. was given. Repeated immunologic studies at the end of the first course of treatment were unchanged, but it was decided to go ahead with the lobectomy. Sixteen days after admission the left chest was explored and the involved left upper lobe was removed. Studies by the pathologist showed extensive lung destruction and microscopic changes consistent with *Blastomyces dermatitidis* infection. Four days after starting a third course of vaccine, or the thirteenth postoperative day, the skin test did become positive. It was at this time that the chest plate showed an area of infiltration in the right mid-lung field; the complement fixation test also became positive.

On the nineteenth day after operation desensitization to the blastomycin was started with a 1:10 dilution given every other day for the next three weeks. On the twenty-sixth day after operation potassium iodide therapy was started and continued in the postoperative period.

Very shortly after the appearance of the positive skin test and complement fixation the lung lesions began to subside. On discharge from the hospital forty-nine days after operation the lungs were clear, and the patient had no symptoms.

One month after discharge he was readmitted because of abdominal pain. An appendectomy was done, but a normal appendix was found. There was no evidence of any mycotic infection. A wound infection developed and was drained by his local physician. This cleared in a very short time.

His last visit to the outpatient department was on Feb. 24, 1953. The patient had epigastric distress typical of a duodenal ulcer. This was proved by x-ray studies. The chest was clear on physical and x-ray examination. Complement fixation and sputum studies were negative.

CASE 2.—J. H., a 41-year-old Negro man, was admitted to the hospital complaining of chest pain, wheezing, and general malaise. Symptoms had begun five months before admission when he noted marked fatigability. One month after the onset of the symptoms he had to stop work and for the first time developed fever. On penicillin therapy he improved for three weeks, but then the symptoms recurred. Then he noticed a cough productive of purulent sputum and severe anterior chest pain. In spite of further treatment with antibiotics the symptoms increased. Careful studies of sputum failed to show any acid-fast organisms. He lost fifteen pounds in this five-month period.

The patient was employed in a woodworking plant and was a part-time barber. Past history and review of systems were noncontributory.

Physical examination showed the blood pressure to be 130/90, pulse 90, respirations of 18, and temperature of 37.6° C. Examination of the chest showed signs of consolidation of the right upper lung field. There was no involvement of the osseous system or genitourinary tract.

Laboratory examination showed the hemoglobin to be 8.9 Gm. White blood count was 24,000 with 80 per cent segmented forms, 6 per cent stabs, 1 per cent eosinophils, 5 per cent monocytes, 4 per cent small lymphocytes, and 4 per cent large lymphocytes. The urine examination was negative. Blood chemistry studies showed the total plasma proteins to be 6.7 Gm. with an albumin of 3.00 Gm. and a globulin of 3.7 Gm.

Smears of the sputum on admission were positive for *Blastomyces dermatitidis,* and this was later confirmed by culture. Skin test to blastomycin six days after admission was positive. Complement fixation studies for blastomyces were negative. X-ray films of the chest showed diffuse consolidation of the right lung with involvement of both the upper and lower lobes (Fig. 2).

Hospital Course.—Eight days after admission a course of blastomyces vaccine therapy for desensitization was started (Chart 2). The initial injection of vaccine was 0.1 c.c., and this was increased by 0.1 c.c. every other day until the sixteenth day after admission when 1 c.c. was given.

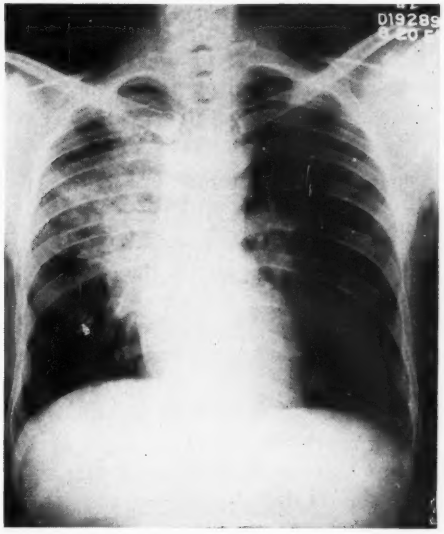

Fig. 2.—Film of patient J. H. with *Blastomyces dermatitidis* infection (Case 2).

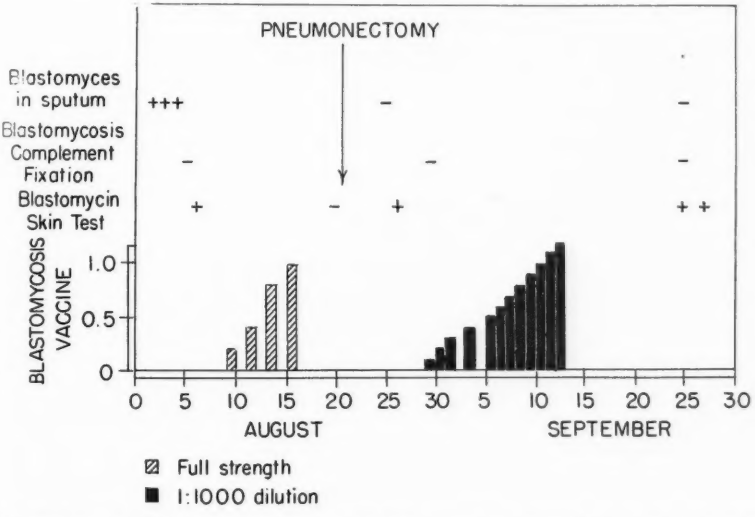

Chart 2.—The chart shows relationship of skin sensitivity, organisms in sputum, and administration of vaccine in Case 2 (J. H.).

On the nineteenth day after admission a right pneumonectomy was performed. The lung showed marked destruction with microscopic changes typical of blastomycosis. On the twenty-sixth day following surgery, the skin test being still positive, desensitization with blastomyces vaccine was started using a 1:1000 dilution. This was continued at home for another month until finally 1.2 c.c. of full strength vaccine was given. After operation chest x-ray examination on the day of discharge showed the right lung to be normal in appearance.

One month after discharge the patient was seen in the outpatient clinic. He had just completed desensitization with blastomyces vaccine, and the skin test was negative. On another visit five months after discharge the skin test was positive.

The patient was last seen in the clinic eighteen months after operation with no complaints. He had gained ten pounds, the sputum was negative to smear and culture, and the left lung was clear.

DISCUSSION

The patients in the two cases presented in this report recovered following the deliberate surgical extirpation of lung infected with blastomycosis. The fortunate outcome in our cases was due to the favorable immunologic response of the patients to the infection. In Case 2, the patient had a positive blastomycin skin reaction and a negative complement fixation reaction to blastomyces. Vaccine therapy was begun to desensitize the patient before surgery, so that there would not occur a hypersensitive reaction to a postoperative spread. The patient's skin test was converted to negative. In view of this response as well as the lack of involvement of other structures, pneumonectomy was done.

The patient in Case 1 on admission had a negative skin test to blastomycin and a negative complement fixation to blastomyces. Surgery in this instance was begun before either became positive. A postoperative spread of the infection occurred and cleared when the complement fixation and skin test became positive. In retrospect the operation should have been deferred until the skin test became positive. The rapid change in the postoperative pulmonary infiltrate was striking and lends further proof to the observation that immunologic findings are sensitive indicators of prognosis.

In 1949, Smith[5] reviewed the immunologic findings in forty blastomycosis patients followed at Duke Hospital. The study correlated the response to treatment and survival rate with the results of the blastomycin skin test and the complement fixation test for blastomyces. This series of forty cases of blastomycosis was found to fall into the following four separate categories:

1. Positive Skin Test with Negative Complement Fixation. This indicates good immunity, and of the ten patients in this category studied by Smith, nine survived.

2. Positive Skin Test and Positive Complement Fixation Test. In this group there is immunity, but the positive complement fixation indicates active disease. In Smith's study of this group of ten there were seven survivors.

3. Negative Skin Test but Positive Complement Fixation. The results in this group were very poor, there being only two survivors in ten cases. The poor prognosis is explained on the advancing active disease indicated by the positive complement fixation associated with anergy indicated by the negative skin test. The anergy is similar to the terminal anergic state in some patients dying from disseminated tuberculosis.

4. Negative Skin Test and Negative Complement Fixation. In nearly every instance this indicates, according to Smith, disease of short duration. There were eight survivors in ten cases of Smith's belonging to this category.

As outlined, the clear understanding of the immunologic responses of the patient to the blastomycosis infection governs the therapy with blastomycosis vaccine and iodide. It may be necessary to use the vaccine to promote immunity in some situations, or to desensitize the patient in other cases. Iodide therapy can be of benefit in the treatment of the disease, but the skin test and complement fixation reaction must govern its use. If iodide is administered to a patient with a low level of immunity, dissemination of the disease, as emphasized by Smith, may occur. The positive skin test and complement fixation are indications of immunity. On the other hand, hypersensitivity to blastomycosis may lead to difficulty with iodide due to dissemination of the products of the blastomycosis infection in a hypersensitive patient. These considerations lead to therapeutic regimes that may appear paradoxical as in Case 1. It may be necessary to use vaccine to incite immunity that at the same time may induce hypersensitivity. Then before giving iodide to the patient with induced hypersensitivity the desensitization with the same vaccine will have to be performed.

Iodide was used in one of the two cases recorded in this report. It was started in Case 1 in the postoperative period only after the patient had shown signs of immunity and then had been desensitized. In Case 2 the patient's progress was so good that it was never deemed worth while to use iodide.

The introduction of therapy with stilbamidine in blastomycosis has altered greatly the treatment of the disease.[4] In the generalized form that is almost uniformly fatal, this therapy has, in our hands, been successful. In eight such cases recovery has occurred, and there has been no relapse over a short follow-up period. Unfortunately there is associated with this treatment a high incidence of trigeminal neuropathy. This has developed in six of our eight cases. Schoenbach, Miller, and Long[4] note and discuss this complication in their paper. In certain selected cases where only the lungs were involved and there was adequate immunity as indicated by a positive skin test to blastomycin, one might consider resection alone. The stilbamidine could be saved for use if complications developed. Pulmonary resection may have to be done eventually in stilbamidine controlled cases, particularly if there occurs extensive lung destruction.

CONCLUSION

Two cases of *Blastomyces dermatitidis* infection of the lung treated by surgical excision are reported. The correlation between the good results and immunologic response of the patients is presented.

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BRONCHIAL RESECTION AND ANASTOMOSIS

Report of a Case

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MUCH interesting work has been done in the field of reconstructive tracheobronchial surgery in the last few years. Many points, however, need more precision and further study. This is the reason I have reported this case, in the hope that it may add a little to the knowledge of the question.

CASE REPORT

E. B., a 52-year-old white man, a barber by trade, was admitted to this hospital on Jan. 15, 1951. Past history revealed that two of his brothers had died of pulmonary tuberculosis. At the age of 30 he had been hospitalized for a pulmonary ailment of an unknown nature. Since then he had been subject to attacks of flu with fever and coughing. In November, 1945, after an especially severe bout of flu, he presented himself at one of the tuberculous diagnostic centers. A cavity was found at the apex of the left lung. He was sent home to rest while waiting for a place at the sanatorium. He was finally admitted in February, 1946. Strict bed rest was enforced until August of the same year. At that time the cavity had disappeared, the basal sedimentation rate was at 4 mm., the sputum and gastric contents contained no tubercle bacilli. General condition was good, but the physician in charge of the case at that time noted an insufficiency of the respiratory function. The patient was discharged with a diagnosis of moderately advanced, nonbacillary, inactive, ulcerofibrous tuberculosis.

Rest was continued at home for four months. After this time he went back to work. Gradually he felt more and more tired and noticed that the cough was increasing in intensity, and the sputum in quantity.

In September, 1950, he had to stop work and stayed at home until re-admission to this hospital, in January, 1951. Upon admission he was very thin and weak, the basal sedimentation rate had gone up to 45 mm., the hemoglobin had come down to 75 per cent. The x-ray examination revealed a cavity at the left apex, and a bronchiogenic spread in both lung areas. Sputum examination revealed a high content of tubercle bacilli. Bed rest was again started, but this time with the addition of 1 Gm. of streptomycin daily. Gradually the lung fields cleared, but the cavity in the upper left apex increased in size. In July, a pneumoperitoneum was started, but had to be discontinued in October, because of gastric distress. A bronchoscopy, performed in December, revealed a normal right bronchial tree. On the left side, a stenosis of the main bronchus was noted, secretions appearing through a pinhole in the center of the constricted area.

A repeat bronchoscopy in January, 1951, revealed an identical picture.

Tomography revealed no cavity on the right side. The electrocardiogram revealed a sinus arrhythmia, and a myocardium of doubtful integrity.

On February 14, the patient was presented at the joint medical-surgical meeting for study. He appeared as a thin man, older in appearance than in years, with palpable radial and temporal arteriosclerotic arteries. It was quite apparent, although complete respiratory function studies were not performed, that this man was in very great need of every bit of normal lung tissue available. He wanted something done to correct his present status. He

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was accepted for surgery, preferably for resection (due to the bronchial stenosis and the two cavities in both the upper lobe and the apical segment of the lower lobe). (Figs. 1 and 2.)

On Feb. 8, 1952, the left chest was entered through the bed of the resected fifth rib. It was soon apparent that the whole upper lobe and the apical segment of the lower lobe had to be resected, due to their gross involvement in disease. This was done quite easily by dissection technique. The remaining segments of the lower lobe appeared perfectly normal and contained no palpable nodules. We were now in this situation: The diseased tissue had been removed, but the stenosis remained between normal bronchus and normal lung tissue (Fig. 3). At it was strongly felt that this particular patient needed the four basal segments, it was decided to attempt a resection of the stenotic area, with end-to-end anastomosis of the bronchus.

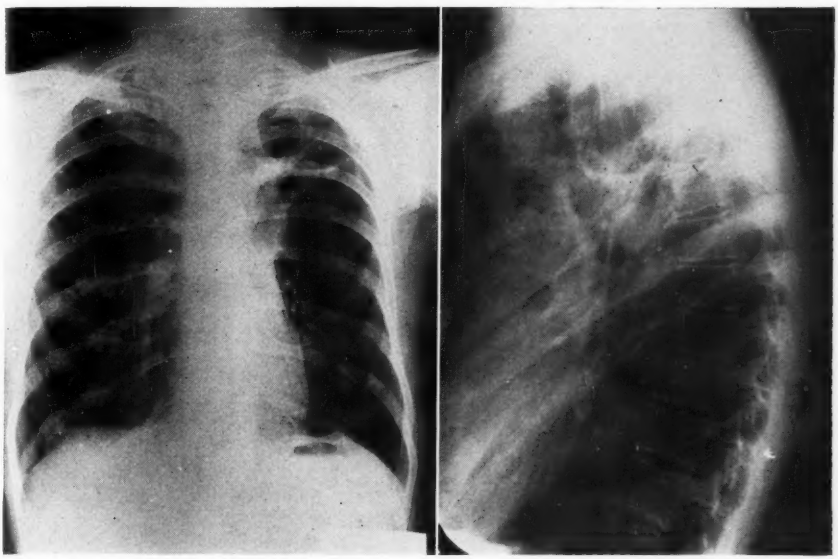

Fig. 1. Fig. 2.

Fig. 1.—Anteroposterior view taken on Jan. 14, 1952. Two different fluid levels can be seen at the left apex. To each of these belongs a specific annular contour.

Fig. 2.—Left lateral view taken on the same day. It shows very well the two distinct annular contours. One belonging to the upper lobe, the other to the apical segment of the lower lobe.

The main bronchus was transected, leaving about a 1 cm. stump. The lower lobe bronchus was next amputated, just below the orifice of the apical segment (Fig. 4).

The lower lobe was then brought up to the main bronchus without difficulty, severing a few strands of the pulmonary ligament helped. The pulmonary artery had to arch its extra length, but there was no acute kinking. The difference in diameter of the two bronchial orifices was not as great as expected. The anastomosis, end-to-end type, simple, non-everting, was easily accomplished; first a few stay sutures of stainless steel were applied and then interrupted silk stitches completed the anastomosis. The anastomosis was completely wrapped in a pedicled pleural flap (Fig. 5).

As soon as positive pressure was applied, the lobe expanded very well. The bronchus was tested for leaks and none were found. The chest was closed in the usual manner, and two drainage tubes were inserted, one anteriorly in the second interspace, and one in the ninth interspace. The patient was returned to his room in excellent condition. Fig. 6 shows the x-ray picture taken on the second postoperative day.

The left lower lobe was expanding well.

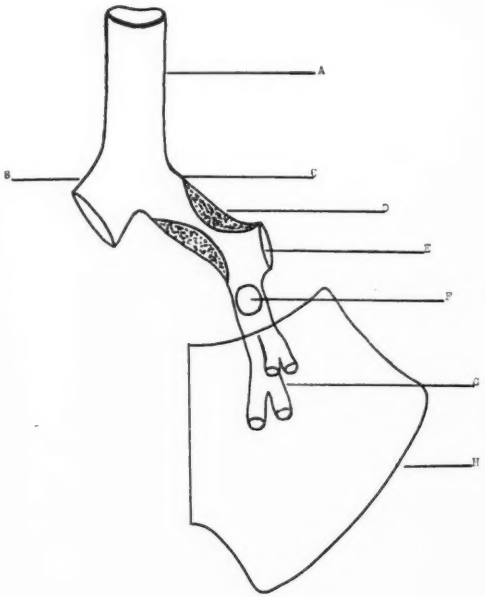

Fig. 3.

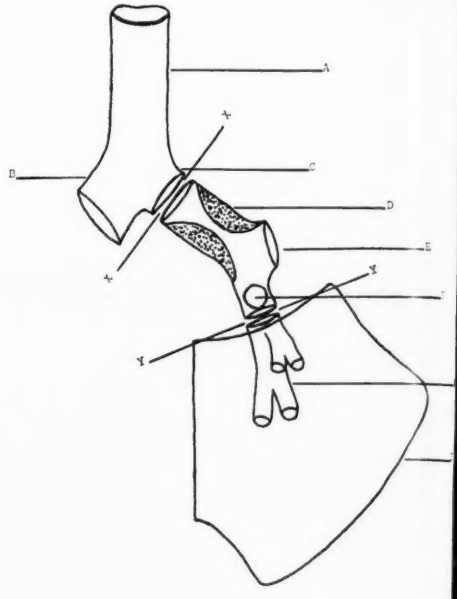

Fig. 4.

Fig. 3.—*A*—trachea, *B*—right main bronchus, *C*—left main bronchus, *D*—stenotic area, *E*—orifice of resected left upper lobe, *F*—orifice of resected apical segment of left lower lobe, *G*—four basal bronchi, *H*—normal lung tissue.

Fig. 4.—*A*—trachea, *B*—right main bronchus, *C*—left main bronchus, *D*—stenotic area, *E*—orifice of resected upper lobe, *F*—orifice of resected apical segment, *G*—four basal segments, *H*—normal lung tissue, *XX*—line of section, *YY*—line of section.

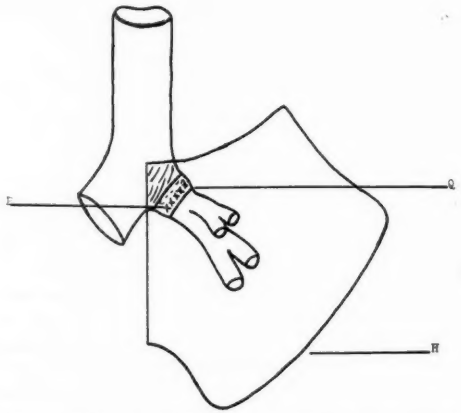

Fig. 5.—*Q*—line of anastomosis, *P*—covering pleural flap.

Difficulty in expectorating was soon noted, and quite a few bedside bronchial aspirations had to be performed. Otherwise the postoperative course was normal.

By the sixth postoperative day (Fig. 7) there was fairly good lung expansion, and the breath sounds were becoming clear and normal.

The lower drainage tube was removed after seventy-two hours. The upper tube was left in for eight days, in the event that a leak might occur in the anastomosis. However, there was no sign of this at any time.

One month after operation, 150 c.c. of clear fluid was removed from the upper chest. This was negative for all organisms including tubercle bacillus.

By March 24, the patient had gained weight, could expectorate easily, and there were very few secretions. Temperature was normal. A first sputum examination was negative. A week later this was positive.

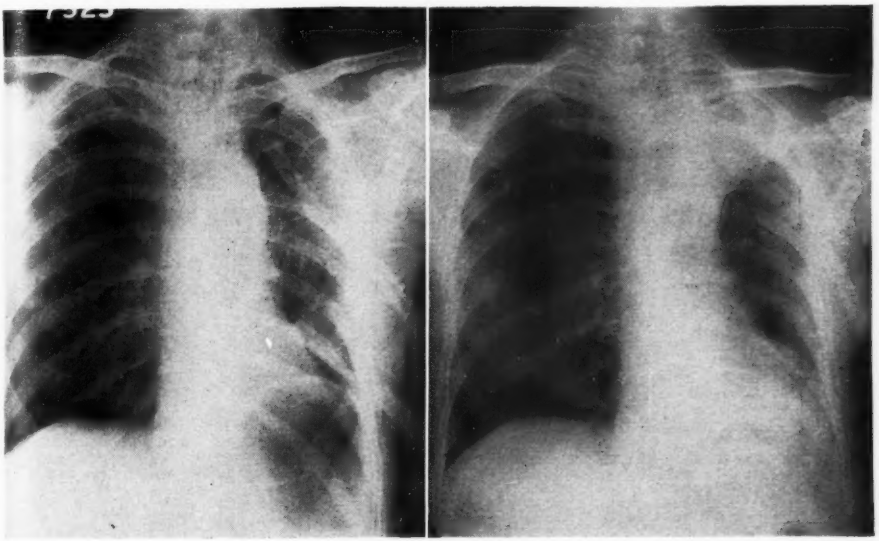

Fig. 6. Fig. 7.

Fig. 6.—X-ray film taken on the second postoperative day.
Fig. 7.—X-ray appearance on the sixth postoperative day.

The roentgenogram shown in Fig. 8 was taken two months after the operation; at this time the basal sedimentation rate was 28 mm.

Bronchoscopy at this time revealed a very well-healed anastomosis and a slight narrowing of two of the basilar segments. Bronchial secretions were negative for Koch's bacillus. Isonicotinic acid* therapy was instituted. The patient then had a vital capacity of 2,100 c.c. Fig. 9 shows the x-ray appearance of the patient three months postoperatively.

The patient, after the operation, had many complaints. He soon learned that he had had a special type of operation. He became very expansive about his condition and, although he was doing well, cried at least once a day. We learned later that he was suffering from a lack of whisky and beer which had been the main items of diet for the last twenty years. However, one complaint made us wonder: the patient complained of pain "in the region of the anastomosis." We could find no reason to explain this.

On June 13, he coughed up a small amount of red blood.

*Rimifon, which was graciously supplied by Hoffmann-La Roche.

On June 16, he complained more intensely of this thoracic pain, and coughed up 50 c.c. of bright red blood. A tomogram was taken at this time to see if it would shed light on whatever process was going on (Fig. 10).

We were thinking of an exploratory thoracotomy when, on June 19, the patient had a massive hemoptysis and died. An extract from the post-mortem report is included here.

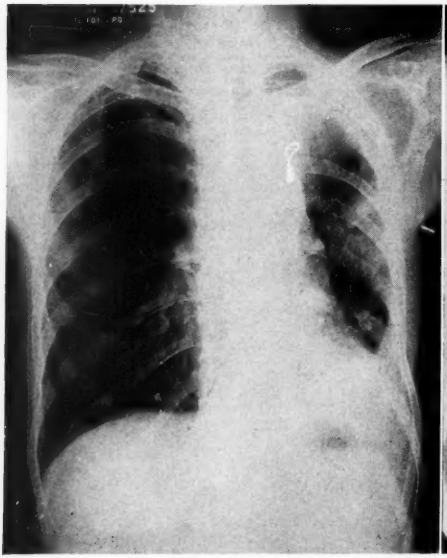

Fig. 8.

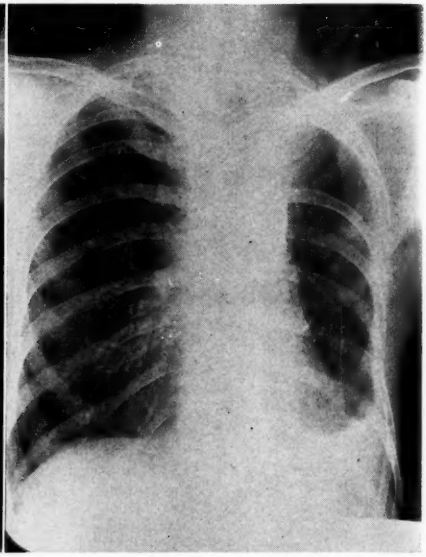

Fig. 9.

Fig. 8.—X-ray appearance two months after the operation with a fair quantity of normally aerating lung tissue on the left. There is a left paratracheal shadow which was taken to be part of the normal healing process.

Fig. 9.—X-ray appearance three months postoperatively.

Thorax.—

Right Side: A few adhesions were present between the apex and the parietal pleura and between the base and the diaphragm. Grossly, the lung was normal. Histologically, the lung contained a few tuberculous micronodules in a progressively fibrotic stage, a moderate anthracosis, and a purulent bronchiolitis.

Left Side: The apex was fixed to the thoracic wall by a very dense and thick fibrous process. A scalpel had to be used for cleavage. There existed to the left of the arch of the aorta, between that structure and the lung surface, a hard fibrotic mass. This extended from the thoracic vault to the lower lobe and was very adherent to the mediastinum and to the superior and posterior parts of the aorta. The center of this mass contained about 5 c.c. of thick pus. (This later was reported as sterile.) The lung itself, reduced to two-fifths of its original size, was red, firm, and presented, in the region of the apical segment, an organizing hematoma. This seemed to have infiltrated its way into the lung substance.

Trachea and Bronchi.—These were filled with blood. The bronchial anastomosis was perfectly well healed and presented a normal mucosa. There was slight narrowing of the lumen.

The Aorta.—Presence of a few atheromatous plaques. In the posterior region of the arch, the aorta is very adherent to the vertebral bodies. Its wall at this level seems to be

split in different layers, and this is in close association with the fibrous mass already described, and with the hematoma in the surface of the lung. An inflammatory process shows the destruction of the elastic medial layer of the aorta.

The examination of the other organs was noncontributory.

Post-Mortem Diagnosis.—Aortic aneurysm ruptured into the surface of the segmented lower lobe of the left lung was the diagnosis.

Fig. 10.—A round, dense shadow appears to the left of the trachea, immediately above the bronchus. This was believed to be a hematoma. 6 cent. tomographic cut.

DISCUSSION

This case certainly brings up the question which concerns a number of the patients, discharged from sanatoriums after medical treatment, who show up later with progressive disease. It seems that about 30 per cent of these patients eventually return for further treatment. This shows the necessity of active therapy in more and more cases. At the time of this patient's first discharge from the hospital, tomograms were not in use, and no cavity could be seen on the plain x-ray film, and there were no clinical signs of activity. Now there is no excuse for discharging a patient, without a tomographic study of the lungs, at least in the suspicious areas.

We have often thought that this patient should have had a tracheotomy at the time of the operation. It would have aided greatly in the postoperative care. Whether it would have influenced the final outcome is impossible to say.

We probably should have been more astute in the selection of this patient for this type of operation, but it must be admitted that he presented all the absolute

indications for this procedure. We have not come to any conclusion regarding the formation and rupture of this aortic aneurysm. We are ready to accept the blame in the line of faulty technique, but we have reviewed this time and again and do not see that we could do better at another opportunity. Possibly we would try to cover the raw area left by the eradication of the apical segment. Can this be incriminated as a causative factor? It is possible, but we do not believe very probable, unless the vessels were already diseased.

At the time of operation we had noted nothing irregular about the appearance of the aorta.

We believe, that in such an operation, a marked inflammatory reaction is inevitable, and if it occurs alongside normal vessels, all will be well, but if it occurs close to an already pathologic aorta, this may be fatal.

CONCLUSIONS

1. Resection and end-to-end anastomosis of bronchi for tuberculous stenosis is a fairly easy procedure, and must be superior to grafts and prostheses.

2. Tracheotomy should be employed as part of the postoperative therapy.

3. Cases should be selected if possible; better results will probably be obtained in younger people.

4. Great care should be exercised in the technique in order not to let vascular structures come into contact with raw areas.

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DILATATION OF MAJOR AZYGOS VEIN SIMULATING A MEDIASTINAL TUMOR

A Case Report

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THERE has been an increasing interest in the significance of mediastinal masses in the past several years, due no doubt to the more frequent use of chest roentgenograms coupled with the greater safety of exploratory thoracotomy. It would seem appropriate, therefore, to report a case of dilation of the azygos vein which simulated a mediastinal tumor on the chest x-ray film.

CASE REPORT

Mrs. M. R., Case Number 10410, a married woman 51 years of age, was admitted to Swedish Hospital on March 7, 1951, with the following history:

In January, 1951, a routine chest x-ray picture taken by the mobile unit revealed a shadow in the right upper mediastinum. She had no complaints and had always been in excellent health. She had had four normal pregnancies. The children were all alive and ranged in ages from 12 to 31 years of age. One sister had died of tuberculosis.

Physical examination was negative except for marked obesity. The patient's weight was 205 pounds; she was 64 inches tall. She had a hypertension of 190 mm. of mercury systolic and 110 mm. of mercury diastolic. X-ray photographs taken by her local physician in the posteroanterior, lateral, and oblique views revealed a shadow of uniform density, having a smooth margin located just to the right of the mediastinum at the level of the major azygos vein immediately posterior to the trachea. The mass was somewhat pear shaped with the narrow end pointing toward the mediastinum. It did not exhibit expansile pulsations (Figs. 1, 2, and 3).

Preoperative diagnosis was either a cyst or a tumor of lymphoid origin. Exploratory thoracotomy was carried out on March 8, 1951. A long posterolateral segment of the right fifth rib was removed subperiosteally and the pleural space entered through the bed of this rib. The lung appeared entirely normal. The tumor mass was easily visualized in the region of the major azygos vein between the vertebral bodies posteriorly and the trachea anteriorly. It was covered with mediastinal pleura and appeared to be filled with dark fluid. It measured about 5 cm. in width and 6 cm. in length. The mediastinal pleura was dissected from the surface of the cystic mass, and at this point the lesion was found to consist of a marked dilation of the major azygos vein. The vein narrowed down in a funnel-shaped manner at its point of entrance into the superior vena cava. The vein was ligated and divided at this point. An attempt was then made to dissect out the venous branches leading into the aneurysmal-like sac; however, exposure of these vessels was very difficult, and during the course of this dissection the thin-walled venous pouch was ruptured. Bleeding could easily be controlled by digital pressure. The rent was closed with a continuous suture of 00000 arterial silk. During the course of this procedure it was noted that a large clot formed in the lumen of the venous sac. This clot was evacuated. The margin of the dilated vessel was turned inward by mattress sutures until the lumen was completely obliterated. The lung was then re-expanded and three intercostal catheters placed in the chest for drainage. The wound was closed with catgut.

Continuous suction was applied to the three catheters. The postoperative convalescence was uneventful, and the three catheters were removed on the fourth postoperative day. The

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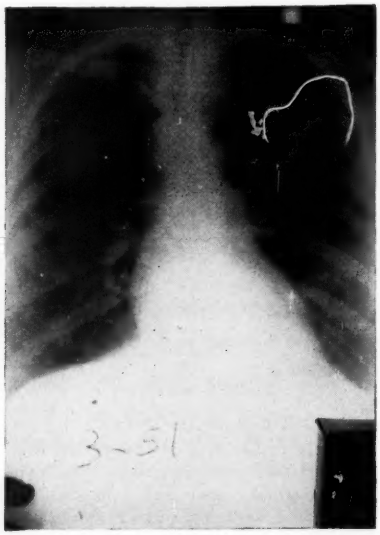

Fig. 1.—Preoperative chest x-ray picture, March, 1951, showing oval shadow of increased density projecting to right of mediastinum opposite the shadow of the aortic nob.

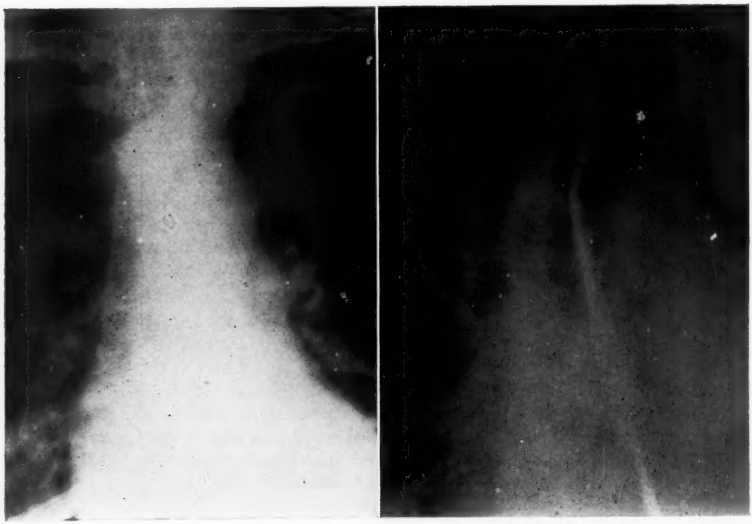

Fig. 2. Fig. 3.

Fig. 2.—Close-up view of lesion seen protruding to right of mediastinum at level of aortic arch (preoperative).

Fig. 3.—Preoperative lateral view of lesion with barium in esophagus.

patient was discharged on March 14. Postoperative chest x-ray films were taken in July, 1951. The shadow which had been present previously can no longer be visualized (Fig. 4). The patient has remained well since her surgery. Studies of the venous pressure in the upper extremities done postoperatively are within normal limits.

ANATOMY

The terminal segment of the major azygos vein can be visualized as a small round shadow producing an indentation on the right posterolateral aspect of the barium-filled esophagus in the right anterior oblique view.[1] The tributaries of the azygos vein may show considerable variation[2]; however, the terminal segment is more constant. Anatomically, the terminal segment of the major azygos vein consists of the portion contained between the point at which the vein leaves the posterior thoracic wall and the point at which it terminates in the vena cava superior.

Fig. 4.—Film taken four months postoperatively, showing absence of mediastinal shadow.

Studies by Andreassi[3] show that the azygos vein leaves the posterior thoracic wall at the level of the inferior portion of the body of the fourth dorsal vertebra. The vein then curves inferiorly to the right, describing a curve with its concavity to the left so as to pass laterally to the esophagus and trachea, and again medially to empty into the superior vena cava. The entrance into the superior vena cava is at a slightly lower level than the point at which it leaves the posterior thoracic wall. As a result of the curved plane, the central portion of the vessel can be demonstrated on x-ray films taken in the usual sagittal projection (the posterior portion by the left anterior oblique projection, and the anterior portion by the right anterior oblique projection).

DISCUSSION

In this case no cause was found to explain the marked increase in size of the azygos vein. Increase in size of the image of the azygos vein has been demonstrated frequently in portal stasis. Grilli[4] has been able to demonstrate significant enlargement of the azygos vein shadow in six out of ten patients with esophageal varices. In the latter condition the azygos vein, along with the esophageal veins, becomes important as part of the collateral circulation. In any cardiac or extracardiac disease that increases the venous pressure in the vena cava superior the azygos vein will enlarge. Durieu and Lequime[5] have demonstrated visible enlargement of the azygos vein in cardiac insufficiency with decrease in the size of the x-ray image following correction of the decompensation. The azygos vein acts as a manometer, signaling the pressures in the vena cava. Variations in size of the image of the azygos vein have been studied and observed in physiologic conditions. An increase occurs during the Valsalva test with changes of body position, in physical exercise, and in phases of cardiac activity.

I can find no reports in the literature in which the terminal segment of the major azygos vein has become dilated so as to cause a roentgen shadow which is of such size as to simulate a mediastinal tumor. It is possible that examination of this mass fluoroscopically with the patient straining, so as to increase the venous pressure, would have demonstrated an appreciable increase in the size of the shadow. This is suggested as a worth-while diagnostic procedure.

Planograms have been of help in separating the image cast by the normal azygos vein from opacities due to tuberculosis[6] and might well have produced valuable information as to the nature of this lesion. Unfortunately, these studies were not done in this case.

SUMMARY

1. A case of abnormal dilation of the terminal segment of the major azygos vein simulating a mediastinal tumor is presented.

2. The various causes of dilation of the azygos vein are discussed.

3. It is suggested that fluoroscopic examination, in an effort to demonstrate changes in size of the mass with straining so as to increase the venous pressure, as well as planographic studies, be carried out in all lesions visualized in this area on the chest roentgenograms.

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NONSPECIFIC LUNG ABSCESS: EXPERIENCE WITH FIFTY-FIVE CONSECUTIVE CASES

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INTRODUCTION

THIS communication records our experience with nonspecific lung abscess during the period 1947 through 1952. Fifty-five consecutive cases are reported; the patients range in age from 20 to 62 years. All of these patients except one were men. This series includes those cases with an insidious onset or in which the pathology was secondary to a clinically recognized, pneumonic infectious process. We were not able to obtain a history of dental or ear, nose, and throat surgery from any of these patients.

No cases secondary to prior disease such as bronchiectasis, carcinoma, adenoma, lung cyst, or tuberculosis are included. Amebic lung abscess, occasionally seen in this area, is not included because of the difference in pathology. No bronchial foreign body cases are included. Three cases previously given adequate treatment for lung abscess at other hospitals and who had uneventful removal of residual lesions are excluded. The cases reported were either proved pathologically (twenty-two) or showed definite radiologic evidence of abscess, such as a thick-walled radiolucency with fluid level or a rounded infiltration not necessarily conforming to a bronchopulmonary segment.

LITERATURE

Reviewing the American literature, Allen and Blackman[1] collected 2,114 cases up to 1936 and found the reported mortality for cases treated by medical methods to be 34.4 per cent and medical plus surgical methods to be 34.2 per cent. Smith[9] collected 2,166 cases reported between 1936 and 1948 and found essentially no change in mortality. Brock,[2] in 1948, emphasized the high mortality rate in lung abscess. He pointed out the danger of embolic infection of other organs, particularly the brain, and the continuous disability resulting from unresolved abscess. He also discussed the factors involved in deciding the appropriate treatment. Other authors[3-8] have reported widely varying experiences, using medical treatment alone, drainage procedures, medical treatment with surgical resection, and various forms of local antibiotic treatment including bronchoscopic instillation.

CLINICAL PICTURE

Classically, lung abscess begins either as a result of an unresolved pulmonary infection with the typical symptoms developing out of the ones already present

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or suddenly in full-blown form, probably as the result of foreign body, aspirated infected material, or embolism. Patients with this condition are supposed to exhibit high fever and cough with production of purulent sputum, which may or may not have a foul odor. Pain and hemoptysis may be present. If the abscess does not connect with a bronchus, there will be much less sputum; however, the breath may be foul regardless.

Even before the widespread use of short courses of antibiotics for pulmonary infections, various observers[2, 5] noted that an occasional patient with this serious condition would show little or nothing in the way of toxicity or presenting symptoms. We have been impressed with the frequency of this situation in the cases seen here in the last three years.

TABLE I. SYMPTOMS

PERIOD	NO. OF CASES	FEVER	CHILL	WT. LOSS	COUGH	CHEST PAIN	HEMOP-TYSIS	FOUL SPUTUM	TOXIC ON ADM.	MINIMAL SYMPTOMS
1947-49	26	23	16	10	23	20	6	20	20	4
1950-52	29	23	7	10	24	24	16	18	12	12
Total	55	46	23	20	47	44	22	38	32	16

Table I shows the symptomatology of our cases, with the cases divided into two nearly equal three-year groups. There was not much difference in kind of symptoms, although there seems to be an increase in hemoptysis. However, the number of cases whose symptoms were minimal, or not indicative of an abscess clinically, has practically tripled; while the number of cases considered toxic (temperature over 101° F., white blood count over 16,000) before start of treatment has decreased to less than one-half (twelve of twenty-nine). We can assume that chronic lung abscess still carries a poor prognosis and remains fraught with secondary dangers if untreated; therefore, any pulmonary symptoms, even if not marked, deserve radiologic examination. Patients with pulmonary infiltrations treated with antibiotics should, of course, be carefully followed until complete radiologic clearing has occurred, as secondary abscesses may thus be treated promptly. Early bronchiogenic tumors presenting as localized pneumonitis may also be identified.

TABLE II. LOCATION OF LESIONS BY LOBE INVOLVED

PERIOD	R.U.L.	R.M.L.	R.L.L.	L.U.L.	LINGULA	L.L.L.	TOTAL
1947-49	6	3	7	5	0	5	26 Abscesses 26 Patients
1950-52	16	4	3	4	0	4	31 Abscesses 29 Patients
Total	22	7	10	9	0	9	57 Abscesses 55 Patients

We have noticed a tendency in the last few years for these lesions to appear in the upper portions of the lung, particularly the right upper lobe (see Table II). The explanation of this is not entirely clear and no illumination on this point is offered in the reviewed literature. Infected material certainly would be more easily aspirated on the right side, but why 40 per cent of

this series should have presented in the right upper lobe is not explained. If this is a true finding, it suggests that patients with acute cavitary, upper lobe lesions, and with sputum free of tubercle bacilli should be subjected to abscess therapy without delay. Fig. 1 shows presenting symptoms in relation to location of disease. One significant finding concerns the large group of right upper lobe cases, although exhibiting about the same amount of pain, seemed to have a lower incidence of foul sputum and initial toxicity than the lesions in other lobes. This probably can be explained mechanically, as an upper lobe lesion should drain well as soon as bronchial communication becomes established.

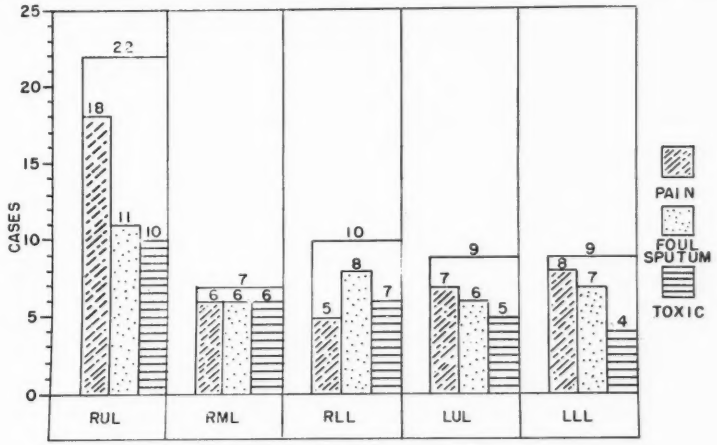

Fig. 1.—Location versus symptoms.

BACTERIOLOGY AND ANTIBACTERIAL TREATMENT

Almost all of these patients had satisfactory cultures made from sputum or bronchoscopic aspiration. Vincent organisms were not routinely looked for by the laboratory. Since they respond readily to penicillin, we have not felt it necessary to request this examination. *Staphylococcus aureus* was consistently found, with *Streptococcus hemolyticus, Pseudomonas aeruginosa, Aerobacter aerogenes, Hemophilus influenzae,* and *Escherichia coli* present less frequently in that order. Fungus cultures have routinely been employed since 1949, but none has been found in conjunction with lung abscess. No matter how long the symptoms of an abscess have been present, antibiotic treatment is instituted to observe what improvement may be obtained. The patients receive at least 1,200,000 units of penicillin per day. Sulfonamides were used in 1947 and 1948 but have not been used for the last four years. If the sputum is not reduced to a small amount, an additional antibiotic is added which is selected on the basis of sensitivity studies. Aerosol penicillin has been used in a few cases, without much additional effect, in our experience.

Bronchoscopy has been of value in ruling out tumors, foreign bodies, and other intrabronchial lesions; and all but a few of these patients were bronchoscoped. Except for temporary aspiration of purulent material, we did not

feel that there was any therapeutic value in bronchoscopy. However, in one case where segmental bronchial constriction was present, bronchial dilatation at intervals was of help in obtaining a cure. It is our opinion after reviewing the resected material that no case not cured by antibiotic treatment would have been cured by bronchoscopic aspiration and antibiotic instillation.

Postural drainage is employed and it is believed to be of benefit depending, of course, on the intelligence and cooperation of the patient.

DURATION OF DISEASE VERSUS NEED FOR SURGERY

Fig. 2 shows the relationship between the duration of symptoms before starting intensive treatment and the eventual need for surgery. As would be expected, the long-standing, fibrous-walled lesions usually required surgical treatment. After six weeks the value of medical treatment decreased greatly; however, it is felt that antibiotics should be used until no further improvement clinically or radiologically is obtained.

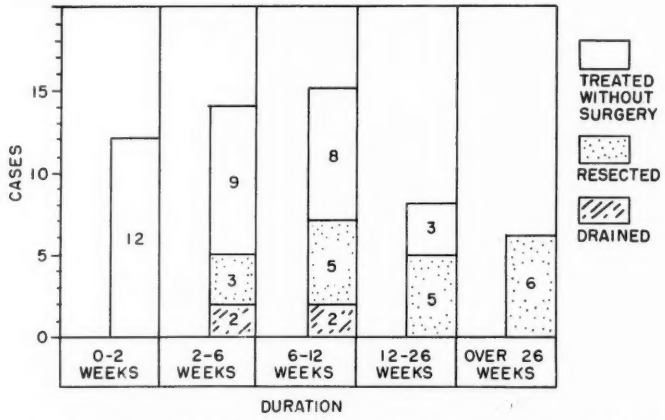

Fig. 2.—Duration of symptoms at the beginning of treatment versus the need for eventual surgery.

Some patients with long-standing disease were cured by the antibiotic agents; and surgery, when required, was easier with the inflammatory reaction at a minimum. Cooperative management between medical and surgical specialists is obviously necessary if fatal outcomes are to be avoided and chronic suppurative situations, difficult to treat surgically, prevented. Bronchoscopy, aside from its diagnostic value, may help in localizing the process and directing surgical treatment. If there is suspicion that an acute abscess is associated with bronchiectasis, bronchography may be justified, although we do not favor it when pneumonitis is present. Small residuals may be evaluated by bronchography, and here it is most useful.

OPERATIVE PROCEDURES

Table III shows the operations performed for lung abscess. A few drainage procedures for acute lung abscess were done several years ago but none has been necessary recently. Primary drainage for acute abscess should not be

TABLE III. TYPE OF SURGERY PERFORMED

PERIOD	NO. OF CASES	DRAINAGE	PNEUMONECTOMY	LOBECTOMY	SEGMENT OR WEDGE
1947-49	12	3	3	4	2
1950-52	11	1	0	6	4

forgotten, but may rarely be needed in the future. Pneumonectomy for destroyed lung resulting from an abscess has been necessary in three patients. Earlier treatment might have made such extensive resections unnecessary, as all of these patients had long-standing symptoms when first seen. Our aim has been to remove as little functioning lung tissue as possible when resecting the disease.

RESULTS

Results are shown in Table IV. Of thirty-two patients treated without surgery, twenty-eight are considered fully cured and three improved. Two of the latter were improved at the time of discharge, but have been lost to follow-up; one refused surgery and has minimal residuals.

TABLE IV. RESULTS

	NO. OF CASES	CURED	IMPROVED	DIED
Without surgery	32	28—88%	3—9%	1—3%
With surgery	23	21—92%	1—4%*	1—4%*

*Treated with drainage.

The patient who died was a young man with lobar pneumonia and septicemia who ran a rapid downhill course despite intensive treatment and died in seven days. The chest x-ray films showed gradually progressing pneumonitis. The diagnosis of abscess was considered but only confirmed at post mortem.

Twenty-three surgical cases produced twenty-one cures. In four of these, secondary drainage or thoracoplasty following resection was necessary for complicating empyema or fistula. One patient, who had a primary drainage procedure, was considered improved when discharged with a draining sinus. He has been lost to follow-up. One patient, an elderly man with senile dementia also treated by drainage, died after a long downhill course.

SUMMARY

1. Fifty-five cases of nonspecific lung abscess are reported.

2. Thirty-two patients were treated with intensive antibiotic therapy alone. Twenty-eight patients were cured, three were improved, and one died.

3. Twenty-three patients required surgery in addition to antibiotic treatment. Resection of the lesion was done in nineteen patients and primary drainage in four. All resected cases and two of the cases having primary drainage were cured. One patient treated by drainage was improved and one died.

4. The symptomatology is discussed with emphasis on the increased incidence of patients demonstrating minimal symptoms since the advent of widespread antibiotic usage.

5. The value of early intensive antibiotic therapy is demonstrated.

6. The value of intensive antibiotic therapy in all cases regardless of duration of symptoms is discussed.

7. Bronchoscopy was considered to be essential in the diagnosis of these cases.

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MEDIASTINAL LIPOMA

The Successful Removal of An Eight Pound, One Ounce (3,657 Grams) Tumor

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SINCE the first case of intrathoracic lipoma reported by Fothergill[1] in 1783, a number of surveys of the literature tabulating the total number of cases have been reported. The first comprehensive review of thoracic lipomas was reported by Heuer[2] in 1933. He described three types of thoracic lipomas. In the first group are the "dumbbell" type lipomas with intrathoracic and extrathoracic tumors connected by an isthmus through an intercostal space; in the second group are those which extend into the neck from the superior mediastinum; and in the third group are those limited to the thoracic cavity.

The most recent review of the literature is that of Keeley, Gumbiner, Guzauskus, and Rooney[3] in March, 1953, who state that they have collected a total of fifty-seven cases of intrathoracic lipoma, and add one of their own, making a total of fifty-eight cases. The largest tumor recorded was found at autopsy to weigh 17 pounds, 6 ounces.

Watson and Urban[4] in 1944 reported the successful removal of a 6.8 pound mediastinal lipoma, and stated that this was the largest mediastinal lipoma which had been excised successfully from the chest cavity and reported in the medical literature until that time.

CASE REPORT

Mrs. L. E., a 42-year-old housewife, was referred to the St. Joseph Hospital by Dr. Claud Bays, Sept. 26, 1950, because a chest x-ray film taken by a mobile tuberculosis unit had shown "fluid in the left chest." She had noticed gradually increasing dyspnea on exertion over a period of about ten years. Eight years prior to admission to the hospital she had to give up swimming because she could swim only ten or fifteen yards before becoming short of breath. At the time of admission to the hospital she would become quite dyspneic after walking up a flight of stairs. She had also noted slight difficulty in swallowing for the past two years.

The patient had had osteomyelitis of the right femur at the age of 6 years, which had resulted in shortening and deformity of the extremity. She could walk with the aid of a crutch, but not very rapidly.

Physical examination showed a well-developed, well-nourished white woman who did not appear to be dyspneic at bed rest. Abnormal physical findings were confined to the chest and the right lower extremity. Examination of the chest revealed marked displacement of the trachea and the heart to the right. The apex impulse was at the right sternal border. The breath sounds and voice sounds were normal over the right lung. There was no dullness to percussion on the right side except over the displaced heart. There was dullness to percussion over the entire left anterior, lateral, and lower half of the posterior chest. Breath sounds and voice sounds were absent over the same areas. Breath and voice sounds were well

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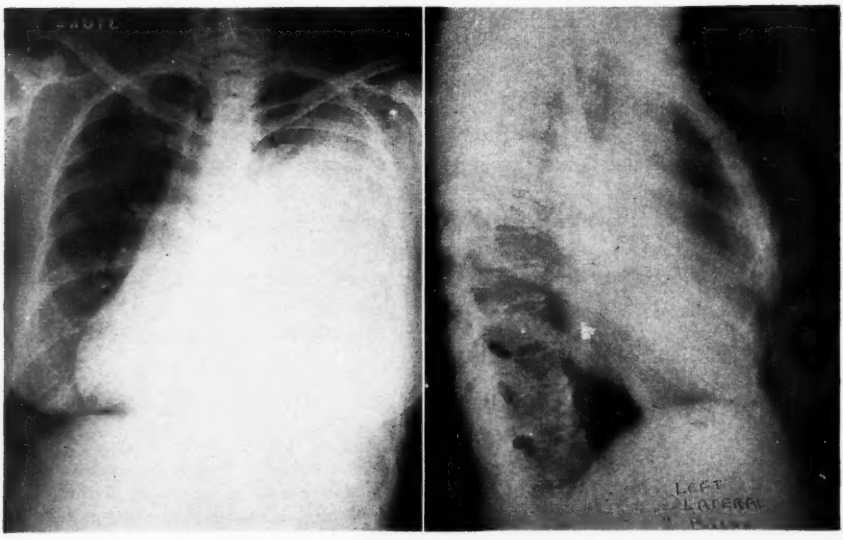
transmitted at the extreme apex of the left chest and over the upper half of the left posterior chest. The left upper posterior chest was resonant to percussion. The heart sounds were of good quality, the rhythm was regular, there were no murmurs; the pulse rate was 84 per minute, the blood pressure was 126/96. There was no paradoxical pulse. The red blood count was 5,010,000; the hemoglobin was 15.5 Gm.; the white blood count was 8,700 with a normal differential count. The urinalysis was negative.

Chest fluoroscopy and roentgenograms in the posteroanterior view showed the left hemithorax to be completely obscured by a homogeneous density which extended upward to the second anterior rib. (Fig. 1.) The lateral view showed the area of increased density to be more marked in the anterior portion of the chest. The posteroanterior roentgenogram showed the density to be less marked at the periphery than at the center. Thoracentesis through the left seventh intercostal space in the anterior axillary line with a No. 13 gauge needle yielded a small quantity of fatty tissue. No fluid could be obtained.



A. *B.*

Fig. 1.—*A* and *B*, Preoperative posteroanterior and left lateral chest films showing the large lipoma of the mediastinum protruding into the left chest.

A presumptive diagnosis of lipoma of the left chest was made because of the long duration of symptoms, the characteristic x-ray appearance (diminished density at the periphery of the mass), and the aspiration of fatty tissue by thoracentesis.

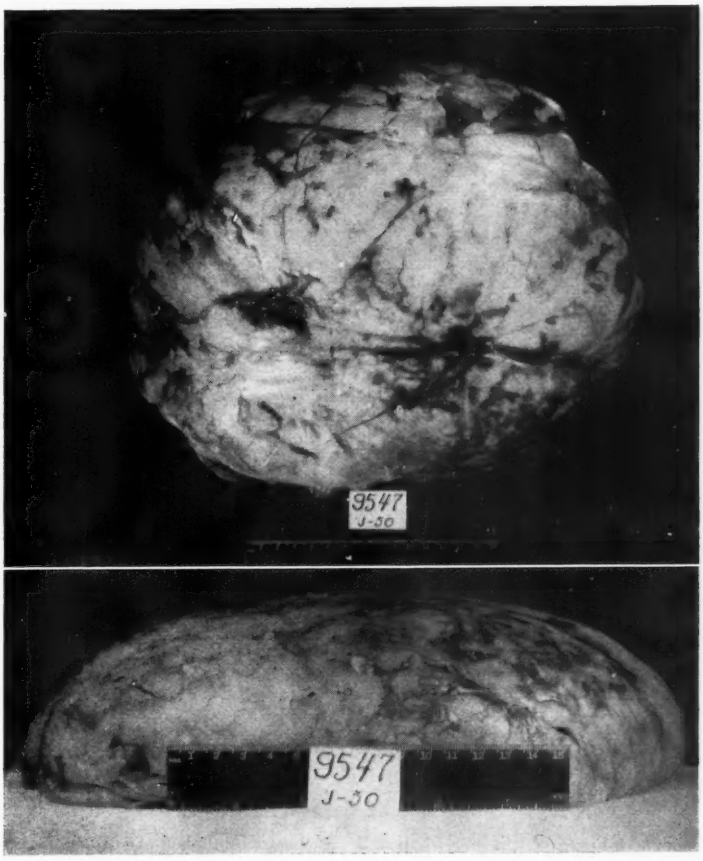
Operation.—On Sept. 28, 1950, the operation was performed. Anesthesia used was Pentothal Sodium induction, cyclopropane, ether administered via an endotracheal tube with assisted respirations.* The patient was then placed in the lateral position on the right side. The left pleural cavity was entered through the bed of the seventh rib, which was excised subperiosteally. On opening the pleura an enormous fatty tumor was seen filling almost the entire left pleural cavity and pushing the mediastinum and heart into the right chest. The mediastinal pleura covered the tumor which was attached to the anterior mediastinum by a broad pedicle. The mediastinal pleura was opened and the tumor enucleated from the mediastinal sac.

The tumor was supplied by one medium-sized artery from the anterior mediastinum. The redundant mediastinal pleura was excised and the defect partially closed, leaving an opening

*Anesthesia was administered by Dr. John Owen.

for drainage into the pleural cavity. Controlled respiration by the anesthesiologist was difficult until the tumor was shelled out of the mediastinal sac. There was a drop in blood pressure at this point from 150/90 to 120/75. The pulse slowed from 100 to 90. There was no other perceptible change in the patient's condition. The left lung when completely expanded appeared to be normally formed in all respects except for its small size. The inflated lung occupied approximately 50 per cent of the left pleural space. There was no exudate over the visceral pleura to prevent complete expansion of the lung. Anterior and posterolateral tubes were inserted and attached to subaqueous drainage bottles before closing the chest.

A.



B.

Fig. 2.—*A* and *B*, Photographs—showing two views of the large, well-encapsulated, lobulated lipoma. The tumor weighed 8 pounds, 1 ounce (3,657 grams).

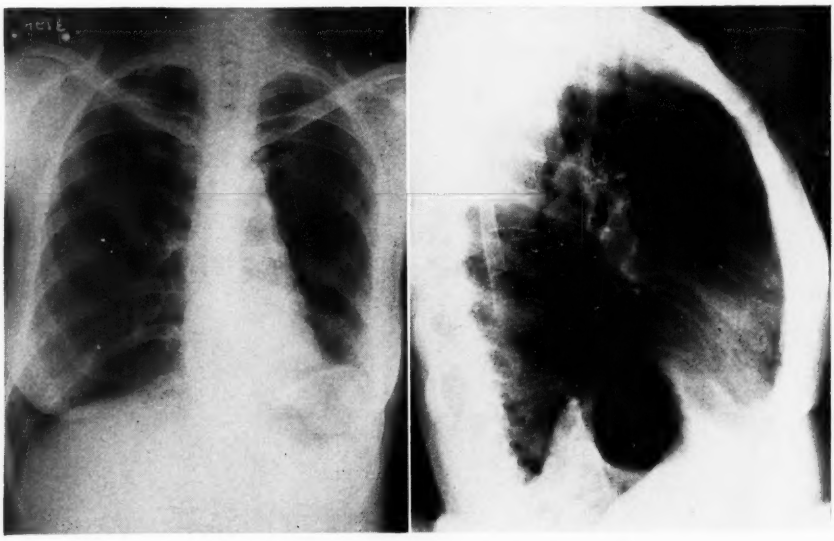
*Pathologic Examination.**—Gross examination revealed the specimen to consist of a large fatty tumor mass which weighed 8 pounds, 1 ounce (3,657 grams), and measured 27 by 24 by 14 cm. (Fig. 2.) Sections through the large mass showed it to be composed entirely

*The pathologic examination was performed by Dr. J. T. McCellan.

of adipose tissue. The histopathology sections showed the structure to be practically normal adipose tissue cells, and the diagnosis was large lipoma from left anterior mediastinum and left chest.

Course.—The postoperative course was uncomplicated except for delayed expansion of the left lung. The intercostal catheters were removed in forty-eight hours. On October 6, thoracentesis was done with removal of 850 c.c. of blood-tinged fluid from the left chest, and three days later 900 c.c. of amber fluid was removed. Thoracenteses were continued at weekly intervals for one month, with removal of about 500 c.c. of amber fluid each time. The patient was then followed with fluoroscopic examination for several weeks. The fluid showed no increase beyond a certain point so thoracenteses were discontinued. It was thought that the fluid was simply filling the space in the pleural cavity which the small lung could not fill.

Subsequent films have shown gradual clearing of the left base. The last film on March 23, 1953, two and one-half years since operation, shows the chest is essentially normal except for slight elevation of the left diaphragm. (Fig. 3.) The patient at this time has no symptoms referable to the chest, except a feeling of slight "tightness" of the left lower chest. There is no dyspnea or difficulty in swallowing.



A. *B.*

Fig. 3.—*A* and *B*, Postoperative films taken two and one-half years after resection of the tumor.

DISCUSSION

The relative infrequency of this tumor in the mediastinum has been emphasized by Blades,[5] who in 1941 reported only two cases of mediastinal lipoma had been seen at Barnes Hospital in St. Louis, and by Sabiston and Scott,[6] who in 1952 reported 101 primary tumors or cysts of the mediastinum observed at Johns Hopkins Hospital from 1933 to 1951. Only two lipomas were included in this series, both originating in the posterior mediastinum. The entirely intrathoracic tumors make up the largest group, forty-one having been recorded at the time of Keeley's report. The most common symptoms reported are those of exertional

dyspnea and the feeling of oppression or heaviness in the chest. The benign nature of the process is suggested by the long duration of symptoms and the good general condition of the patient with the presence of a large intrathoracic mass. The diagnosis prior to operation has been made in only six cases prior to the present report, and was based on aspiration of fat in four cases. Lipoma may be suspected when there is a relatively clear peripheral zone. This has been pointed out by Heuer to be due to the difference in specific gravity between fat and fluid.

The suggestion of Keeley and associates seems appropriate that intrathoracic lipomas be classified in two groups: those that are entirely intrathoracic, and those that have intra- and extrathoracic portions. The latter group is more frequently discovered by the presence of the extrathoracic portion, whereas the intrathoracic types are usually discovered during routine x-ray examination of the chest, or because of the cardiorespiratory symptoms due to the location and size of the tumor.

It has been pointed out by several authors that there may be compensatory adjustments of the cardiorespiratory system due to the large space-occupying mass. The sudden removal of the mass may produce serious cardiorespiratory problems.

The most common postoperative complication, as exemplified by the present case report, has been the accumulation of serosanguineous fluid. It seems plausible that the increased negative pressure in the pleural cavity after removal of the large mass with insufficient lung volume to fill the cavity is probably the most important factor in the production of the fluid.

SUMMARY

A report of the successful removal of an 8 pound, 1 ounce mediastinal lipoma is presented. It is believed to be the largest intrathoracic lipoma surgically removed to this date.

This is the seventh intrathoracic lipoma which has been diagnosed prior to operation. Five of these, including the present case, were based on aspiration of fat.

The present case makes a total of fifty-nine cases of intrathoracic lipomas reported, of which thirty-one have been successfully removed.

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USE OF THE THORACOSCOPE IN THE DIAGNOSIS OF CERTAIN INTRATHORACIC NEOPLASMS

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AT TIMES the physician is confronted with the problem presented by the case in which he feels certain malignant disease is present, but in which he is unable to establish a tissue diagnosis. In some cases the criteria of inoperability, such as extensive spread or metastasis, are already present or would be if a definite histologic diagnosis could be made. This is especially so in thoracic surgery, where often the diagnosis of malignancy can only be made at the time of open thoracotomy. In many such cases bronchoscopy, bronchial washings, needle biopsies, and supraclavicular fat pad biopsies are of no avail, and if malignancy could be proved in some other way the patient could be spared the morbidity and expense of exploratory thoracotomy for an obviously inoperable lesion.

In certain selected cases of intrathoracic malignancy, especially when pleural effusion is present, thoracoscopy with biopsy of suspicious pleural nodules or plaques may be of aid in establishing the histologic diagnosis. Direct visualization of the pleural surface for pleural adhesions was introduced by Jacobaeus in 1922,[1] after ten years of experience with the method. Chandler and Morlock[2] in 1938 first described the use of thoracoscopic biopsy in the diagnosis of various intrathoracic lesions. Since then there have been only occasional remarks in the liteurature concerning this little used but sometimes very valuable adjunct to the surgeon's armanentarium.[3-5] The procedure of thoracoscopy is simple to perform, tolerated well by the patient, and can be carried out in any center where a thoracoscope is present.

TECHNIQUE

The instrument in use at this hospital is a standard Coryllos thoracoscope with two sheaths (Fig. 1). The patient is placed in the supine position or with the affected side slightly elevated. The procedure is carried out under local anesthesia, the thoracoscopic trocar and sheath being inserted through the most appropriate interspace.

After entering the pleural cavity the trocar is removed from the sheath, and pneumothorax is thus established. All fluid is removed from the pleural cavity by insertion of a small catheter through the sheath and the application of suction. The telescope and light are then introduced and the pleural cavity inspected. The diaphragm, visceral and mediastinal pleural surfaces can be well visualized, and adhesions, nodules, fluid, or other abnormalities can be easily seen and studied.

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If suspicious nodules can be seen, especially on the diaphragmatic or mediastinal surfaces, a second trocar and sheath are inserted through an adjacent interspace. Through the second sheath an appropriate forceps can be inserted and biopsies taken under direct vision. It has been our policy to biopsy areas of shaggy fibrinous exudate on the pleural surfaces even if these do not appear suspicious of tumor, and on several occasions nests of malignant tumor cells have thus been found. Direct biopsy of the intact and apparently normal visceral pleura is not advisable, however, because of the possibility of perpetuating the pneumothorax.

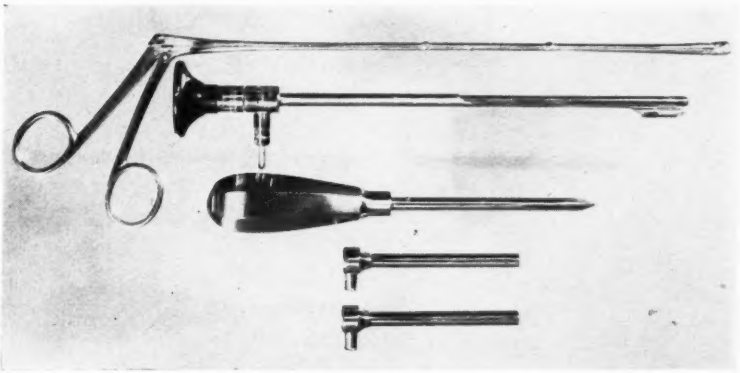

Fig. 1.—The thoracoscope with double sheath and laryngeal biopsy forceps.

Upon completion of the examination a small catheter is inserted into the pleural cavity through one of the trocars (usually the most superior one), and after the removal of the trocars the lung is re-expanded by suction through the catheter. After re-expansion of the lung, the catheter is removed. If it is desired to maintain constant negative pressure for a more prolonged period a No. 18 Foley catheter may be inserted through the sheath instead, the bag of the catheter inflated, and the catheter attached to an underwater-seal drainage bottle. All of our cases have withstood the procedure without incident.

CASE REPORTS

The following are illustrative case reports of five *consecutive* patients undergoing thoracoscopy with biopsy at this institution. A diagnosis of malignancy was made on histologic study in all cases. Four patients were saved the necessity of an open thoracotomy, and would have undergone such had the thoracoscopy been unrevealing. Subsequent autopsy was not permitted on three of these patients and the sole basis for the correct diagnosis lay in the results of the thoracoscopic biopsy.

CASE 1.—T. McC., No. 48506. This 60-year-old patient entered the San Francisco Veterans Administration Hospital on Jan. 15, 1952, complaining of pain in the right chest, dyspnea, cough, and a six pound weight loss during the past two months. His local physician had performed two right thoracenteses. On entry the patient appeared dyspneic, anemic, and somewhat icteric. There was evidence of pleural fluid up to the fourth rib anteriorly.

The left chest was clear. There was palpable enlargement of the liver. Chest x-ray pictures taken on entry revealed massive pleural effusion on the right to the level of the third rib anteriorly. In addition, there was a rounded density 2 cm. in diameter visible in the right upper lung field, which was thought to be either a pulmonary tumor or loculated fluid. Bronchoscopy on Jan. 18, 1952, revealed displacement of the coryna (tracheal carina) to the left, but no visible endobronchial neoplasm. Microscopic study of the sediment from several thoracenteses revealed no abnormal cells. On Jan. 25, 1952, a right supraclavicular fat pad biopsy was done and one lymph node was obtained which was reported to show reactive hyperplasia.

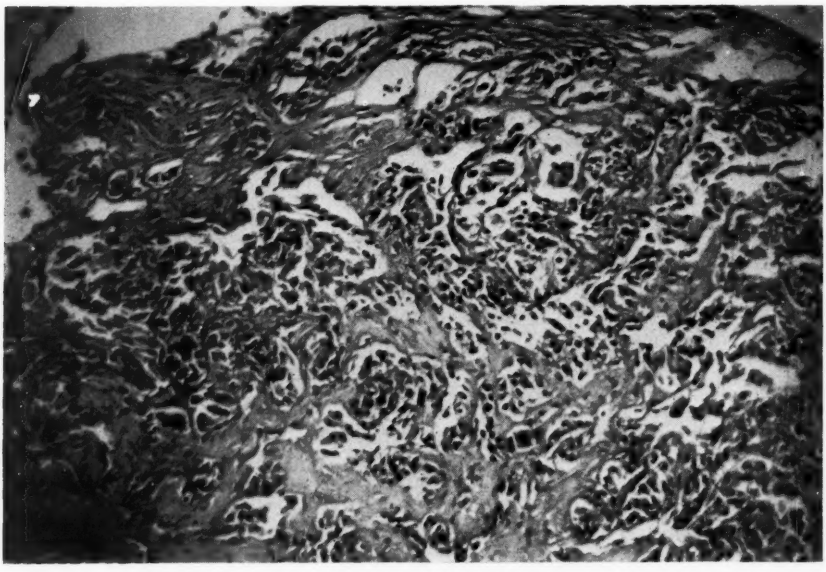

Fig. 2.—Photomicrograph (×115) from Case 1 demonstrating nests of malignant cells in a pleural plaque. This is representative of the biopsies obtained in the other reported cases.

On Feb. 1, 1952, thoracoscopy was performed under local anesthesia, and numerous small nodules were seen on the anteromedial surface of the lung and on the lower parietal pleura. Biopsies taken through a second sheath demonstrated "carcinoma of parietal pleura" (Fig. 2). The patient was considered to have inoperable carcinoma of the lung, was given appropriate supportive care, gradually "went downhill," and expired on March 27, 1952. Autopsy was not permitted.

Case 2.—F. E. B., No. 49253. This 58-year-old man entered San Francisco Veterans Administration Hospital on Feb. 17, 1952, because of increasing dyspnea, cough, and malaise of six months' duration. He had lost five pounds of weight in the three weeks prior to entry. Examination on entry revealed diminished expansion of the left chest, flatness to percussion, and decreased breath sounds and tactile fremitus over the entire left chest. The cardiac border was not discernible. Chest x-ray films revealed a massive left pleural effusion. Culture of pleural fluid on entry was positive for pneumococci. Repeated thoracenteses were done between February 18 and March 5, all yielding about 1,000 c.c. of serosanguineous fluid. Chest x-ray pictures taken at all times revealed only massive pleural effusion. Cytologic studies on the pleural fluid were inconclusive. Bronchoscopy undertaken on March 3, 1953, revealed edema and narrowing of the orifices of the left upper and lower lobe bronchi but no intrinsic tumor.

Thoracoscopy was done on March 5, 1952, at which time numerous gray-white plaques of tissue were seen on the diaphragmatic pleura. Biopsy of these through a second sheath was reported by the pathologist as demonstrating "undifferentiated malignant tumor." On this basis thoracotomy was not performed. The patient gradually became weaker and expired on March 26, 1952. Autopsy was not permitted.

CASE 3.—J. E. I., No. 52578. This 58-year-old white man entered the San Francisco Veterans Administration Hospital on July 7, 1952, complaining of shortness of breath and hemoptysis of three weeks' duration. Physical examination on entry revealed a markedly wasted patient who was lethargic but in no acute distress. There was tracheal deviation to the right, the right chest was dull to percussion, and breath sounds were absent up to the third interspace anteriorly. A right thoracentesis on July 9, 1952, yielded 1,500 c.c. of bloody fluid which was reported by the pathologist to contain "suspect cells." Bronchoscopy on July 16, 1952, revealed the right main-stem bronchus to be pushed superiorly, and the coryna deflected to the left. No tumor was seen.

It was felt that the patient most likely had inoperable bronchiogenic carcinoma, but in order to obtain proof a right thoracoscopy was undertaken on July 18, 1952. The diaphragm and mediastinal pleura were covered with numerous red-gray nodules. Biopsies of these nodules were taken through a second thoracoscopic sheath. The pathologist's report of the biopsy specimens was "undifferentiated carcinoma." Thoracotomy was not performed, and the patient was transferred back to his local community for terminal care.

CASE 4.—H. L. M., No. 52966. This 58-year-old man entered San Francisco Veterans Administration Hospital on July 21, 1952, complaining of right upper quadrant abdominal pain and low right back pain of one month's duration. The patient had undergone thyroidectomy for enlargement of the thyroid in 1929, and had been told at that time there was no malignancy. Because of progressive thyroid enlargement again he underwent a second thyroidectomy in 1944. At this time a histologic diagnosis of carcinoma of the thyroid was made. A swelling subsequently reappeared in the right lower neck and gradually increased in size. On June 1, 1952, the onset of deep right upper quadrant pain caused him to seek medical care. A gastrointestinal series at this time was negative for peptic ulcer. The pain gradually increased in severity in spite of treatment. About June 15, 1952, the patient developed right chest pain and dyspnea, and massive pleural effusion was diagnosed.

On entry to the hospital he was seen to have a 4 by 4 cm. globular mass in the lower right anterior neck. No other cervical adenopathy was noted. The right chest was flat to percussion, with decreased tactile fremitus, voice, and breath sounds. The liver was firm, nontender, and enlarged to 4 cm. below the right costal margin. There were no other significant findings. A right thoracentesis yielded bloody fluid but cytologic studies failed to demonstrate malignant cells.

A liver biopsy on July 26, 1952, revealed malignant cells in the liver.

Thoracoscopy was done on Aug. 6, 1952, and demonstrated much thick gray fibrinous material on both visceral and parietal pleural surfaces. Biopsies of this fibrinous exudate through a second sheath revealed "adenocarcinoma metastatic in pleura." Excision of the neck nodule was carried out on Aug. 12, 1952, and this did prove to be an adenocarcinoma of the thyroid remnant. The patient subsequently became myxedematous. He was given thyroid-stimulating hormones but no uptake of radioactive iodine by the tumor in the chest or liver could be demonstrated. He gradually became weaker and expired on Sept. 26, 1952. Autopsy was obtained and revealed generalized carcinomatosis due to metastasis from an adenocarcinoma of the thyroid gland.

CASE 5.—J. R., No. 53090. This 60-year-old man entered San Francisco Veterans Administration Hospital on July 25, 1952, because of shortness of breath and left chest pain of two months' duration. He had been treated with digitalis without satisfactory response. A chest x-ray picture was taken, which revealed a left pleural effusion. The patient had lost forty pounds of weight during the four months before admission. Examination on admission revealed a moderately obese patient with moderate dyspnea and hoarseness of voice. There

was a left Horner syndrome. Dullness to percussion was noted on the left chest below the first anterior interspace. The trachea was deviated slightly to the left. A left thoracentesis on July 29, 1952, yielded 700 c.c. of serosanguineous fluid. No malignant or suspicious cells were detected in the thoracentesis fluid.

The patient underwent thoracoscopy on Aug. 1, 1952, at which time it was noted that the pleural surfaces were covered with a shaggy grayish exudate, but no actual tumor nodules were made out. Through a second sheath multiple biopsies of the exudate were taken. The pathologists' report on the biopsies was "undifferentiated carcinoma." On Aug. 2, 1952, the patient had a bout of severe cyanosis and dyspnea from which he spontaneously recovered. He had another bout of cyanosis and dyspnea on Aug. 4, 1952, and expired suddenly. Autopsy revealed carcinoma of the left lung with pleural, hepatic, splenic, renal, cardiac, and rib metastases. The immediate cause of death was a right pulmonary embolism.

COMMENT

Frequently the diagnostic procedures of bronchoscopy, bronchial washings, needle biopsy, and supraclavicular fat pad biopsy are of no avail in the diagnosis of intrathoracic malignancy. Under these circumstances, thoracoscopy may be of great help in establishing a diagnosis.

Thoracoscopic biopsy is a procedure, the value of which may not be realized by many general and thoracic surgeons. In certain selected cases it may provide a proved diagnosis and at the same time establish inoperability of a lesion, and save the patient the morbidity and expense of exploratory thoracotomy.

It is to be emphasized that the procedure is not recommended in cases in which an apparently operable lesion is present, because of the possibility of contamination of the pleural cavity with tumor cells. The procedure may be indicated in certain cases of intrathoracic extrapulmonary tumor, pulmonary malignancy with signs of pleural spread, metastatic tumors, and lymphomas with effusion. The technique is simple. The procedure is well tolerated by the patient and can be carried out by any surgeon with a moderate amount of thoracic surgical experience. Most medical centers, especially those treating tuberculosis, possess the required instrument.

SUMMARY

The indications for thoracoscopy with biopsy and its technique are briefly discussed. The simplicity of the procedure is emphasized. Experience with five consecutive cases of obscure pulmonary disease in which thoracoscopy with biopsy was done is cited. In all of these cases an accurate histologic diagnosis was made.

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HIATUS HERNIA: THE PROBLEM OF DIAGNOSIS

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DIAGNOSIS of hiatus hernia has for years been the responsibility of the radiologist. The clinical impression which could not be substantiated roentgenologically has not ordinarily been considered adequate to justify surgical exploration for confirmation and repair. Diagnosis based on the history and physical examination has been discouraging when the roentgen method has been used as the diagnostic base line. Gastroscopic and esophagoscopic examinations have frequently been made in a search for the complications of hiatus hernia, and the results have raised certain questions about the diagnosis of the basic disease. The present report deals with diagnostic experiences among a group of patients proved by radiologic or endoscopic means to have a hiatus hernia.

MATERIAL AND METHODS

Among adult patients studied because of upper abdominal and chest complaints during the past three years, the diagnosis of hiatus hernia has been made by combined roentgenologic and endoscopic techniques eighty-two times. All lesions were of the direct or common type, with simple transhiatal displacement of cardia and varying amounts of stomach. In twenty-one instances the esophagus was judged to be permanently shortened, the other hernias showing ability to slide. Eleven of the patients were women.

The roentgenologic studies were made by several radiologists, always with the request to search for diaphragmatic hernia. The esophagoscopic and gastroscopic examinations were made by one observer who, in each instance, was aware of the findings of at least one recent roentgenologic examination. The Eder-Hufford esophagoscope, which is fitted with a swinging telescope furnishing four-diameter magnification, was used.

In eight patients, brain clips were fastened to the esophagogastric junction following transesophagoscopic biopsy, by a technique previously described.[1]

RESULTS

Roentgenologic examination was successful in establishing or confirming the diagnosis in 78 per cent of the eighty-two cases, 60 per cent on the first examination and the rest upon repeated study following endoscopic detection of the disease (Table I). A total of 159 x-ray examinations were made, and the lesion was demonstrated during 76 (48 per cent).

Table I shows that esophagoscopy was the most efficient of the diagnostic methods. Of fifty-one patients examined, the diagnosis was established or confirmed in 86 per cent. The first esophagoscopy was positive in 80 per cent. In

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TABLE I. CASES OF PROVED HIATUS HERNIA. DIAGNOSTIC RESULTS OF THREE EXAMINATION METHODS

	X-RAY	ESOPHAGOSCOPY	GASTROSCOPY
No. patients examined	82	51	41
No. patients with positive examination	64	44	19
Total no. examinations made	159	68	56
No. positive examinations	76	57	20
First examination positive: no. patients	49	41	16

those instances in which the radiologist had already detected the hernia, the endoscopist approached his examination forewarned. Of the sixty-eight esophagoscopic examinations 84 per cent were positive.

Gastroscopy was the least efficient diagnostic method. Forty-one patients were examined. The diagnosis was made in 46 per cent. The first examination was positive in 39 per cent.

If the roentgenologic method alone had been relied upon, the diagnosis would have been overlooked in 40 per cent (of eighty-two patients). Esophagoscopy alone would have failed in 20 per cent (of fifty-one patients), and gastroscopy in 61 per cent (of forty-one patients).

TABLE II. ESOPHAGEAL COMPLICATIONS. DIAGNOSTIC FINDINGS AMONG 51 PATIENTS STUDIED BY X-RAY EXAMINATION AND ESOPHAGOSCOPY

	TOTAL NO. DIAGNOSED	NO. DIAGNOSED BY X-RAY	NO. DIAGNOSED BY ESOPHAGOSCOPY
Esophagitis	7	0	7
Stricture	2	2	2
Ulcer	3	1	3
Diverticulum	3	3	0

Diagnosis of the esophageal complications of hiatus hernia (Table II) clearly required both radiologic and esophagoscopic study. The seven instances of esophagitis discovered by endoscopic means were not detected roentgenologically, and only the latter method demonstrated the three cases of esophageal diverticulosis. Two of three esophageal ulcers were found only by esophagoscopic examination. The two strictures were easily diagnosed by both methods.

Gastroscopy proved more helpful than roentgenography in the diagnosis of secondary changes within the herniated portion of the stomach (Table III).

TABLE III. GASTRIC COMPLICATIONS. DIAGNOSTIC FINDINGS AMONG 41 PATIENTS STUDIED BY X-RAY EXAMINATION AND GASTROSCOPY

	TOTAL NO. DIAGNOSED	NO. DIAGNOSED BY X-RAY	NO. DIAGNOSED BY GASTROSCOPY
Erosive gastritis	10	0	10
Ulcer: in hernia	2	0	2
Elsewhere	8	8	6

Parenthetically, it may be mentioned that among these eighty-two patients the clinical studies, as dictated by the individual clinical problems presented, demonstrated nine instances of Saint's triad[2] (hiatus hernia, gallstones, and diverticulosis coli).

In eight patients radiologic-esophagoscopic disagreement aroused particular local controversy, and, therefore, transesophagoscopic biopsies were taken at the

esophagogastric junction and brain clips fastened to the biopsy sites for roentgenologic reference. Five of the biopsy specimens showed only gastric mucosa, one esophageal, and two mucosa from the esophagogastric junction itself. In all instances later fluoroscopic study revealed the clips lying superior to the hiatus. The radiologist was then able to demonstrate a hernia in four patients. Two of the others were treated medically. The other two were operated upon, the hernia demonstrated, and the hiatus repaired.

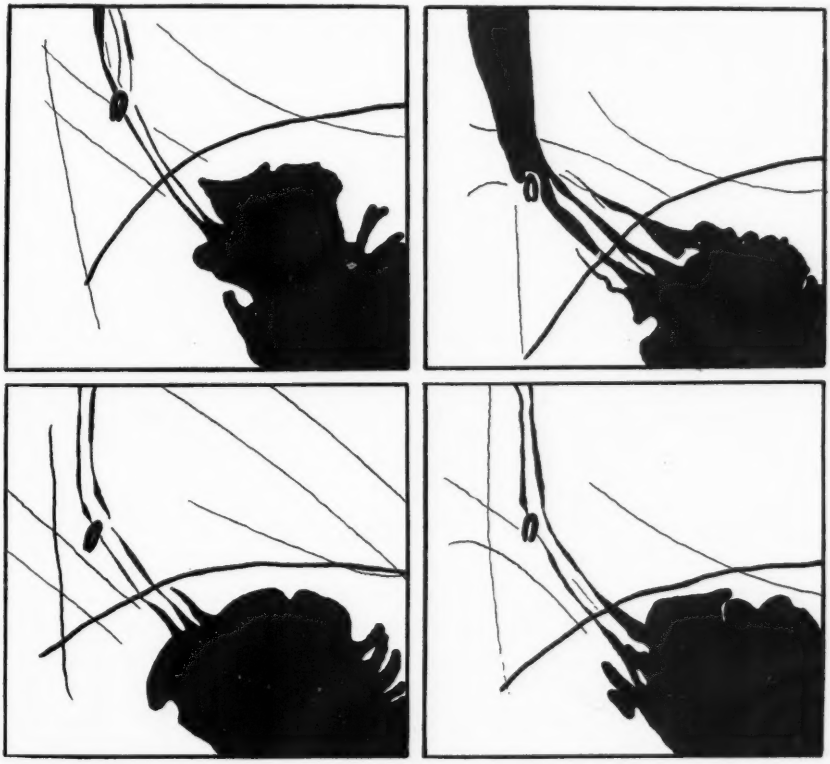

Fig. 1.—(Case 1). Tracings of serial spot films of cardia, made in Trendelenburg's position following attachment of clip to esophagogastric junction. Biopsy at site of clip revealed gastric mucosa. Radiologic evidence of hiatus hernia could not be found. The mucosal pattern distal to the clip seemed to be that of the esophagus. A 4 cm. hiatus hernia was found at operation.

CASE REPORTS

CASE 1.—This 51-year-old white man had complained of burning substernal pain with aggravation upon recumbency for fifteen years. A gall bladder with stones had been removed four years previously, and x-ray studies had demonstrated diverticulosis coli. Two upper gastrointestinal x-ray examinations now were normal, with failure to demonstrate abnormality at the cardia.

Esophagoscopic examination revealed the esophagogastric junction to be approximately 4 cm. above the diaphragm. A biopsy was taken at the junction and brain clips attached to the biopsy site. The specimen was found later to be of gastric mucosa. Immediate fluoroscopic study was unsuccessful in demonstrating a hiatus hernia (Fig. 1).

At thoracotomy a hiatus hernia extending about 4 cm. above the diaphragm was found and repaired.

CASE 2.—A 22-year-old white soldier had complained of daily nausea, epigastric burning, and gas for two years. There was moderate aggravation upon recumbency. Five roentgenologic examinations, with particular reference to the cardia, failed to reveal a diaphragmatic hernia.

Four esophagoscopic examinations were made. A hiatus hernia was found at each, and it was estimated that the esophagogastric junction lay 4 cm. proximal to the hiatus. Biopsies were taken below the junction on two occasions, and both specimens were found to be of gastric mucosa. Clips were placed at the biopsy site the second time, and roentgenologic examination showed the clips to lie about 6 cm. above the hiatus, but no hernia could be demonstrated (Fig. 2).

At thoracotomy the diameter of the hiatus was found to be about twice normal. Six linear centimeters of the stomach extended above the diaphragm. The defect was repaired.

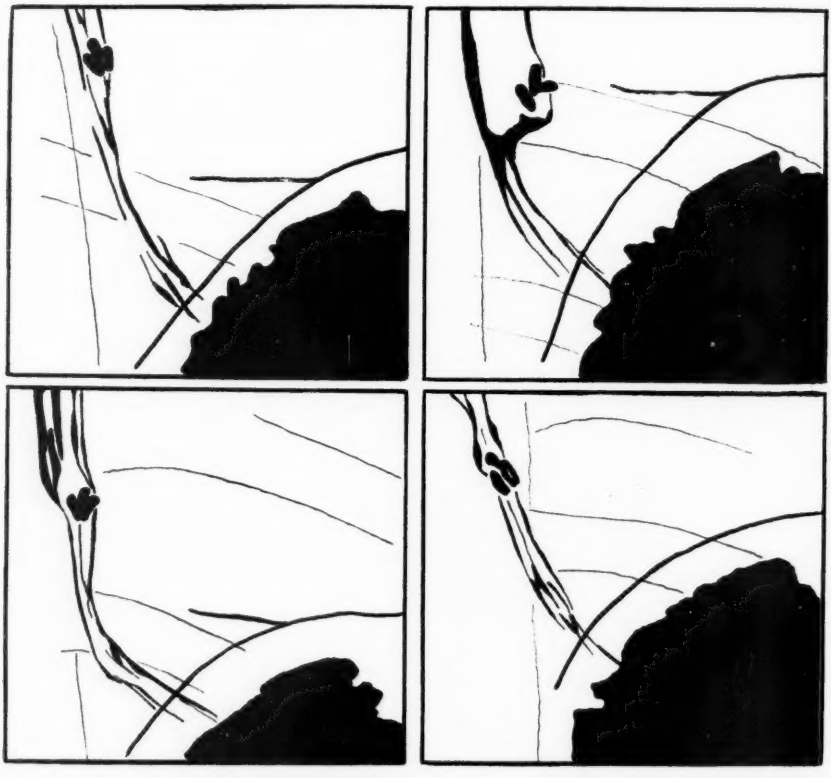

Fig. 2.—(Case 2). Tracings of spot films, as in Fig. 1. Biopsy taken at site of the three clips was of gastric mucosa. The mucosal pattern below the clips seems to be esophageal. Abnormality could not be demonstrated roentgenologically. A 6 cm. hiatus hernia was found at thoracotomy.

COMMENT

The propensity of some hiatus hernias to undergo spontaneous and rapid reduction is only one of the factors hindering diagnosis. The radiologist is not able to detect the junction between esophageal and gastric mucosa and must

render his opinion on the basis of the number and configuration of the regional mucosal folds, plus the point of sudden increase in the tube's diameter. To the esophagoscopist, mucosal appearance is paramount, and, with the help of telescopic magnification, the line marking the esophagogastric mucosal junction is easily recognized in most cases. The number and configuration of regional folds are rapidly changing endoscopic features, and to the esophagoscopist are not particularly useful for differentiation of the two organs. Similarly, true variations in lumen diameter are often difficult to recognize.

The esophagoscopist and radiologist, then, depend on quite different anatomic features for the diagnosis of hiatus hernia. In order to evaluate the diagnostic problem, it is necessary to determine the relationship of the line of the esophagogastric junction to the point at which the digestive tube's lumen suddenly widens. This latter feature has been viewed, classically, as marking the end of the esophagus and beginning of the stomach. However, regardless of gross configurations, any part of the digestive tract which is covered by gastric mucosa contiguous with the mucosa of the stomach, as classically defined, must be considered stomach. The nature of the mucosa makes the difference because, as Barrett[3] has emphasized, any segment covered by gastric mucosa functions physiologically as stomach and falls heir to the diseases of the stomach.

In a previous study[4] it was found that part of the normal resting "abdominal esophagus" is lined by gastric mucosa which may extend as far as 3 cm. upward into the "esophagus." Furthermore, the mucosa of the esophagogastric junction region is capable of automatic rhythmic migration between diaphragmatic hiatus and brink of gastric cavity, possibly through the action of the muscularis mucosae.

These matters may well explain the larger number of hiatus hernias which can be recognized by the esophagoscopist, as observed in the present study and as previously reported by Clerf and associates.[5] The radiologist and esophagoscopist are, in fact, using different definitions of the disease, "hiatus hernia."

Presumably, hiatus hernia is a disease rather than a simple subclinical anomaly because of the gross configurational changes involved, particularly those which destroy the competence of the closing mechanism at the cardia. Although the nature of this mechanism is not at all clear, it is certain that gross shifts in the relationships of the regional organs are much more important than mere misplacement of mucosal types. For this reason, the esophagoscopist's diagnosis of hiatus hernia may at times imply less actual disease than the radiologist's.

The important consequence of an incompetent cardia is retrograde reflux of gastric contents, leading at times to secondary peptic esophagitis, ulcer, stricture, fibrosis, and esophageal shortening. These are complications secondary to damage of the esophageal mucosa. Gastric mucosa in the "abdominal esophagus" is not immune to the influence of regurgitated gastric juice either, and, in fact, Barrett[3] has proposed that many reported instances of esophageal ulcer may actually have represented gastric ulcers in unrecognized proximal extensions of the gastric mucosa.

One interesting implication of Barrett's proposal has to do with the problem of aberrant patches of gastric mucosa which sometimes are found in the mucosa of the distal esophagus. Tissue taken from the edges of esophageal ul-

cers upon occasion shows gastric mucosa, and, consequently, it has been suggested from time to time that aberrant islands of gastric mucosa may play an important role as locus minoris resistentiae for peptic ulcer of the esophagus. This is not the time to discuss aberrant gastric mucosa, but it is interesting to speculate upon possible misinterpretation of contiguous mucosal extensions for isolated islands.

The present study indicated clearly that the esophagoscopic method is able to bring to light a significant number of clinically important, surgically demonstrable hiatus hernias which cannot be recognized upon repeated radiologic study. Use of the biopsy and brain-clip technique has been most instructive in proving the point.

SUMMARY AND CONCLUSIONS

Eighty-two adults with hiatus hernia were studied roentgenologically and endoscopically. The diagnosis was made by repeated roentgenologic examination in 78 per cent; the first examination demonstrated the defect in 60 per cent. Of those examined esophagoscopically, the diagnosis was made in 86 per cent, 80 per cent upon the first examination. Gastroscopic study revealed the hiatus hernia in 46 per cent of those examined, 39 per cent upon the first examination.

All of the examination methods proved indispensable for demonstration of the esophageal and gastric complications of hiatus hernia.

Study of eight patients by means of transesophagoscopic biopsy and attachment of brain clips showed that the roentgenologic method was not effective in distinguishing the mucosal pattern of the esophagus from that of the stomach. It is believed on the basis of previous studies that the gastric mucosa usually extends upward into the normal resting "abdominal esophagus" and that the mucosa of the esophagogastric junction may migrate rhythmically from the level of the diaphragmatic hiatus to the brink of the gastric cavity. Some implications in regard to hiatus hernia are discussed.

Appreciation is expressed to Colonel Sanford W. French, Medical Corps, United States Army and to Lieutenant Colonel John S. Paul, Medical Corps, United States Army for the accomplishment of the thoracic surgery described.

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CIRCULATORY STATUS OF RESECTED TUBERCULOUS PULMONARY LOBES

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OBSERVATIONS have been recorded concerning the vascular supply of the lung since the time of Galen, who is said to have been the first to recognize the double supply of that organ. William Snow Miller's[1] classic monograph on the subject summarized the observations of numerous eighteenth and nineteenth century anatomists and contributed much that still remains the basis for our present knowledge. It is well known that the pulmonary arteries are end arteries. Normally there is no precapillary communication between the pulmonary artery and the bronchial arteries. Communication between these two systems is said to occur only by means of a capillary bed in the walls of the respiratory bronchioles. How much of the blood from the bronchial arteries, if any, normally reaches the walls of the alveoli themselves is not known.

In chronic diseases of the lung changes are known to occur in both of the arterial systems. In 1929, Kaufman[2] described the obliterative changes in the pulmonary artery in tuberculosis, and in 1933, Coryllos[3] speculated on the probable beneficial effect of the changes on the disease. Obliterative endarteritis is a characteristic feature of pulmonary tuberculosis. This was well demonstrated by Birkelo and Brosius[4] in 1938. They injected the pulmonary arterial circulation of tuberculous lungs obtained at autopsy. Their conclusions were that pulmonary tuberculosis causes a marked decrease in pulmonary arterial circulation in the portions of the lung involved and that the impairment was proportional to the degree and stage of the involvement. They also emphasized the *permanency* of these changes.

Frequent references to the changes which occur in the bronchial arteries in chronic pulmonary disease are to be found in the current literature. Liebow and his associates[5] state that in their experience the most striking changes are observed in bronchiectasis. The bronchial arteries usually form a dense plexus of sizable thick-walled, but large-lumened channels and their anastomoses with the pulmonary artery may have a diameter as large as 2 mm. Liebow and his co-workers did not find evidences of precapillary connection between pulmonary arteries and veins as suggested by the observations of Prinzmetal and his co-workers,[6] except in congenital hemangiomas of the lung.

Despite the observations of Harrison and Liebow[7] indicating that a lung deprived of its pulmonary arterial supply may still possess respiratory function, the likelihood of the bronchial circulation's playing a significant role in the

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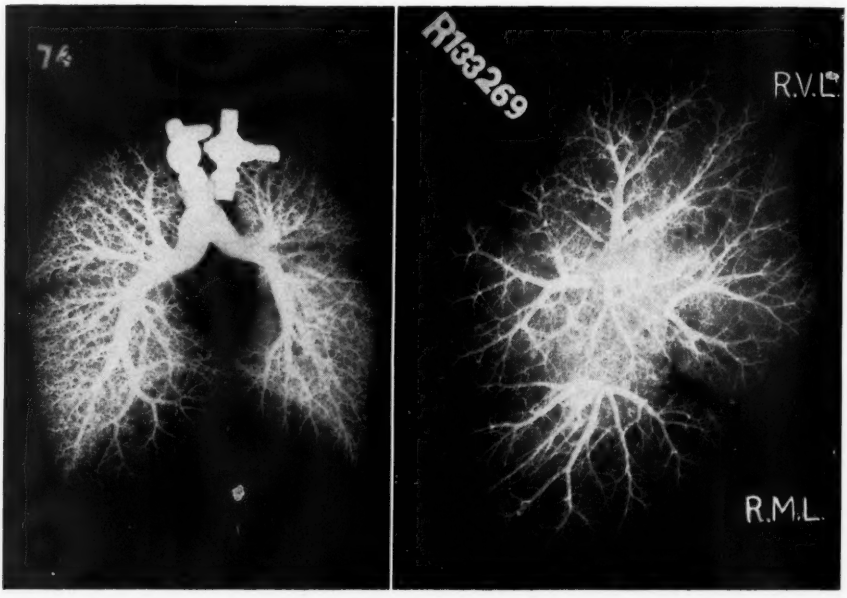
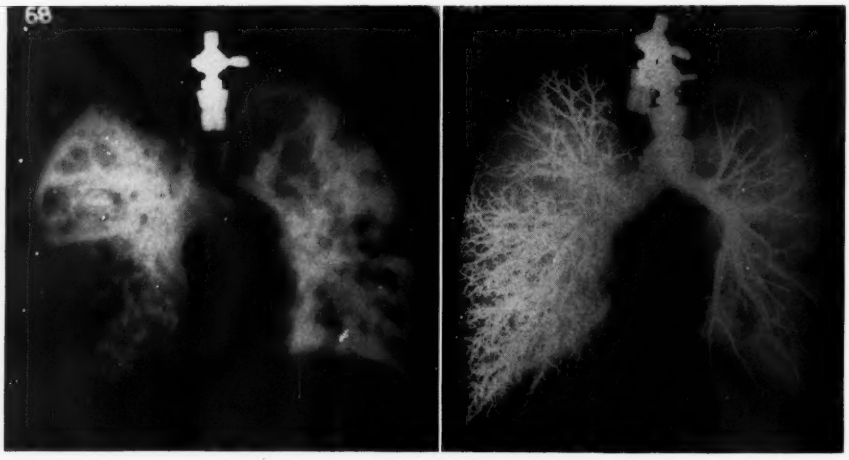


A. *B.*

Fig. 1.—*A*, Injection of normal pair of lungs from a child. Note the uniform filling and graduated decrease in caliber from major vessels to arterioles and precapillary twigs. *B*, Injection of normal right upper and middle lobes from an autopsy on an adult who died of meningitis. There is some emphysema present, particularly in the apex of the upper lobe where blebs are seen.

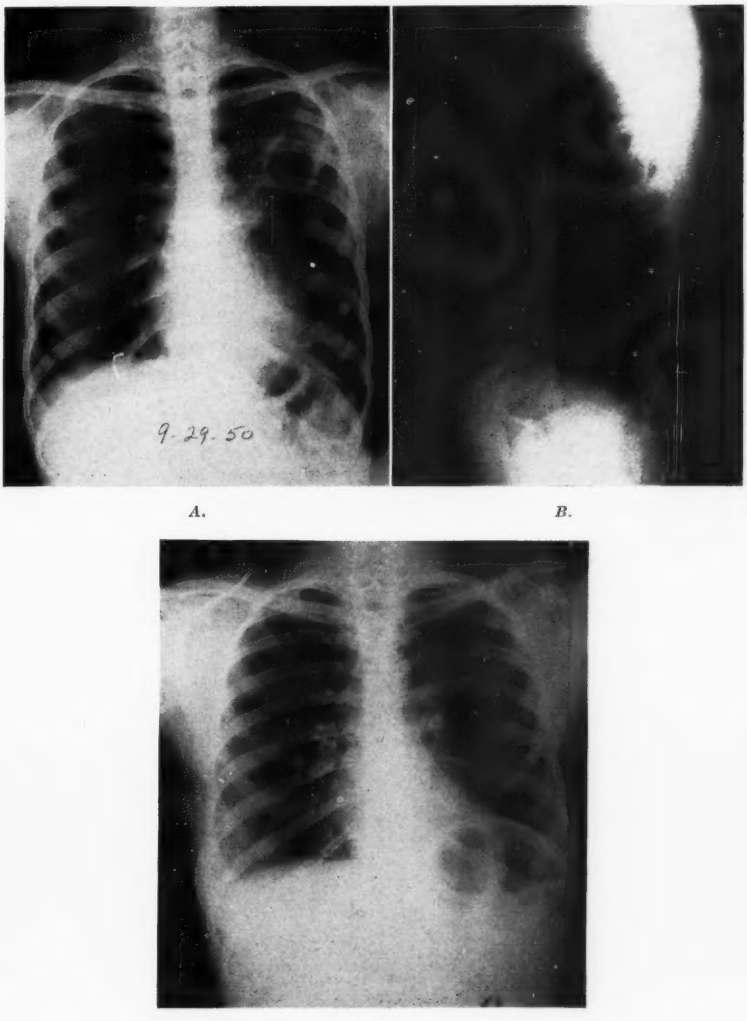


A. *B.*

Fig. 2.—*A*, X-ray picture of an uninjected specimen. The entire left lung is involved in exudative disease while there is also a band of exudative disease in the upper right lung field. There is scattered productive disease throughout the remainder of the lung field. *B*, Injection of the specimen, in *A*, reveals a destroyed circulation in the areas of exudative disease. The vessels are straight, thin, and stripped of their end arteries. A similar involvement has taken place in the area of exudative involvement on the right.

diffusion of gases is rather remote, and for all practical purposes it can be disregarded. For the purpose of this paper we shall exclude entirely the bronchial arterial component.

In the present resection era, in the therapy of tuberculosis, the previously mentioned anatomic changes assume practical importance. There is considerable enthusiam today for segmental and subsegmental resections for residual caseous foci remaining after long-term antibiotic therapy. It occurred to us that, in

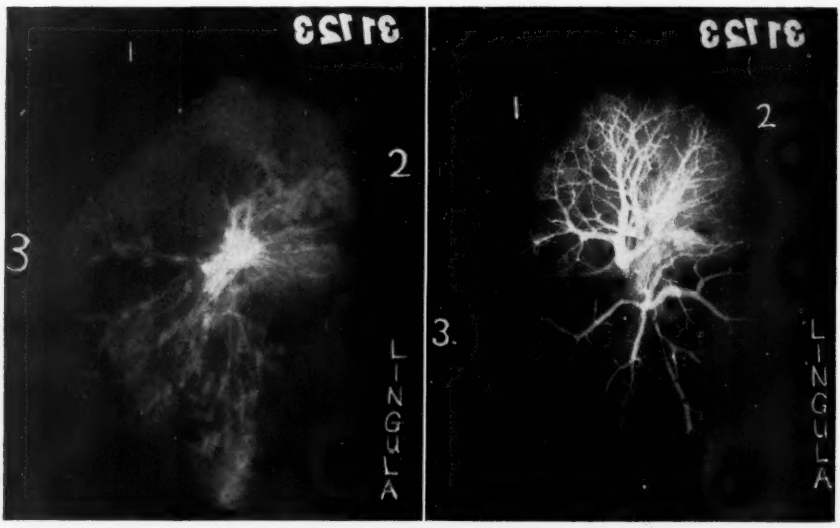


A. *B.*

C.

Fig. 3.—*A* and *B*, X-ray films of a patient taken with disease at the height of involvement. The cavity lies in the anterior segment, as can be seen in the lateral view. There is also involvement of the posterior segment and the lingula. *C*, This x-ray picture was obtained two years later and immediately prior to surgery. It reveals that the cavity has cleared down to a slit and that the remaining infiltrates have cleared extensively.

the extensively involved lobe, which had shown rather striking x-ray regression after therapy, it might be expected to find significant impairment of the vascular pattern throughout the lobe and not only limited to the areas of residual x-ray lesions. If this hypothesis were to prove correct then its implications regarding the functional capacity of the lung would be clear, and lobectomy, rather than segmental or subsegmental resection, would be indicated despite considerable improvement on serial x-ray films. If such a status did not obtain with any degree of consistency then the indication should be clear for limiting the resection to the segments or subsegments containing the residual lesion evident in the x-ray picture.



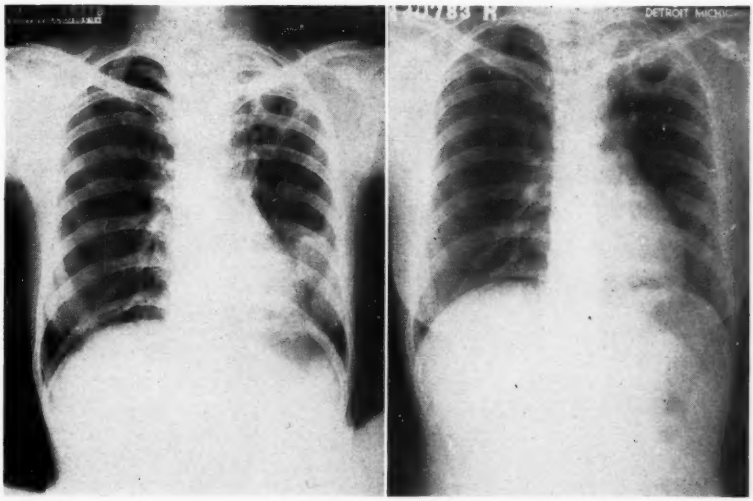
A. *B.*

Fig. 4.—*A*, This is a roentgenogram of the uninjected resected specimen from Fig. 3. The segments are numbered according to the Jackson-Huber classification. Notice that only the apical segment is uninvolved. *B*, This is a roentgenogram of the injected specimen. The apical segment has a normal circulation; as is the case with uninvolved segments, this apical segment has overexpanded and at first glance would appear to represent a considerable amount of functioning lung tissue. In actuality an overdistended, normally vascularized segment will be less efficient than a similar segment of normal volume. The posterior segment shows long thin trunks, crowded together, deficient in branching and end arteries. The underlying luminosities are due to the infiltrations; similar areas are seen in the tip of the lingula. In the anterior segment which was the site of the cavity the vessels fail to inject and lack branches. The lingular vessels are similarly involved. It is apparent that the vascular changes are more consistent with the parenchymal involvement at the height of the disease than that at the time of surgery.

CLINICAL MATERIAL

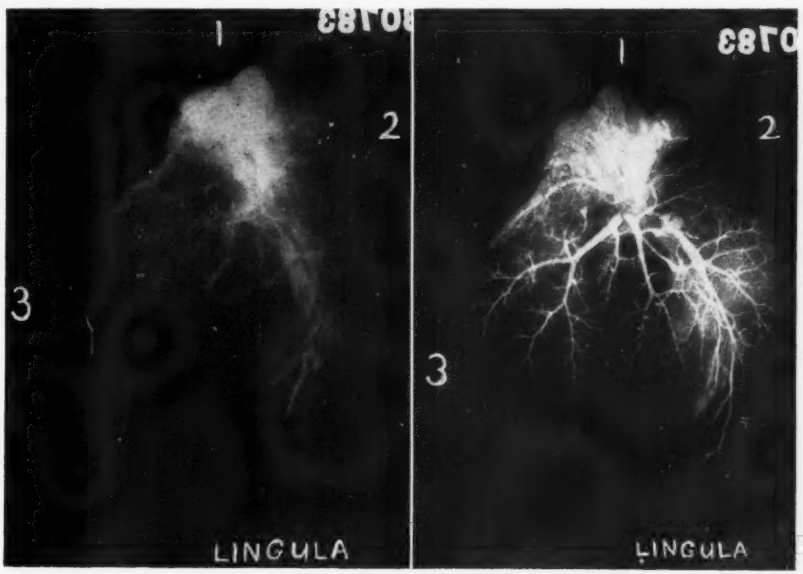
In the course of this study, thirty-five resected lobes have been studied by means of roentgen visualization of the Lipiodol-injected pulmonary arteries. The individual arteries were injected with Lipiodol under a pressure of 25 mm. Hg after the lobe had been inflated with air to a pressure of 4 to 6 cm. of water.

In some of the specimens the lung could not be inflated to its normal size because of pleural tears resulting from difficult dissections. Many of the specimens were rejected because of technical errors in the injection, and others were



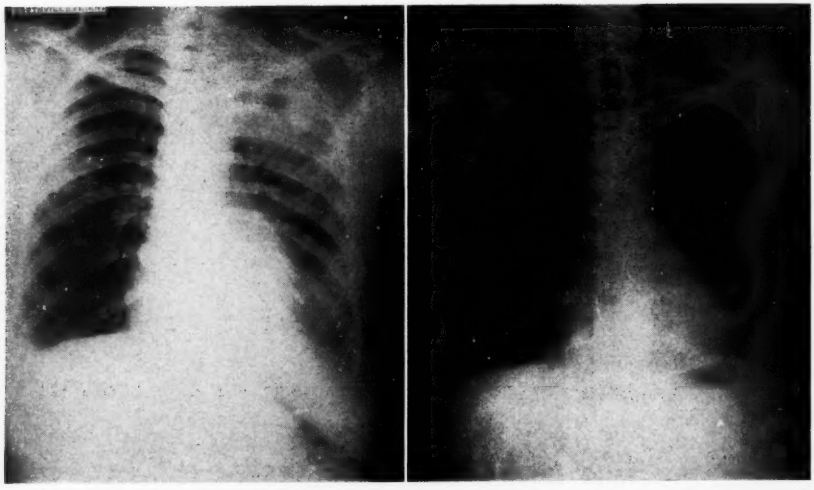
A. *B.*

Fig. 5.—After thirteen months of therapy the infiltrations in the apicoposterior and lingular segments cleared and the 4 cm. cavity closed. Three months later it reopened. *B*, Roentgenogram taken immediately prior to a left upper lobectomy.



A. *B.*

Fig. 6.—*A*, Roentgenogram of the uninjected resected specimen shows complete involvement of the apical segment and a lesser involvement of the posterior and lingular segments. *B*, Roentgenogram of the injected specimen shows a destroyed apical circulation represented only by thin nonbranching stems. The posterior segment shows a lesser degree of the same change and there are involved areas in the upper lingula (area to the left). The anterior segment shows compensatory overexpansion but a relatively normal circulation. It is interesting to speculate upon what function testing of such a lobe would reveal, if it were feasible.



A. *B.*

Fig. 7.—*A* and *B*, Here, there is diffuse involvement of the left upper lobe which, after twenty-three months of therapy, has cleared to an apical cavity.

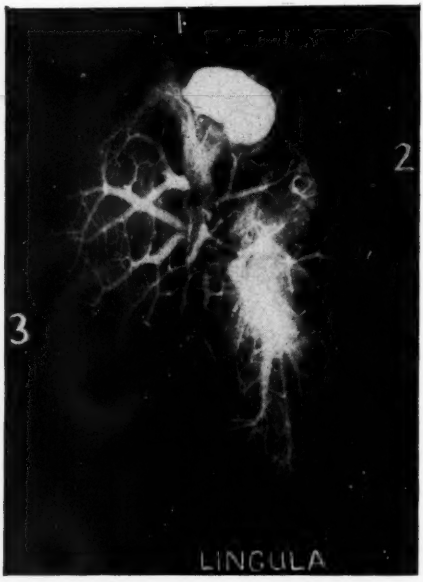
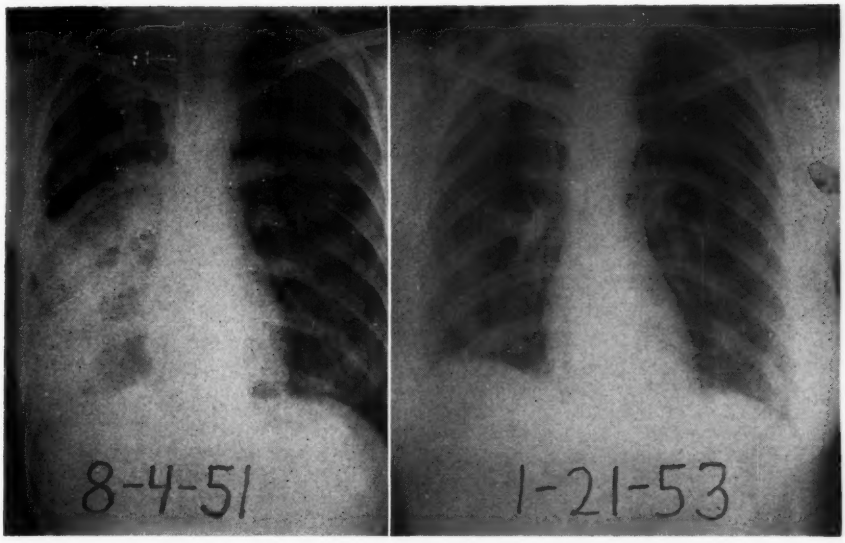


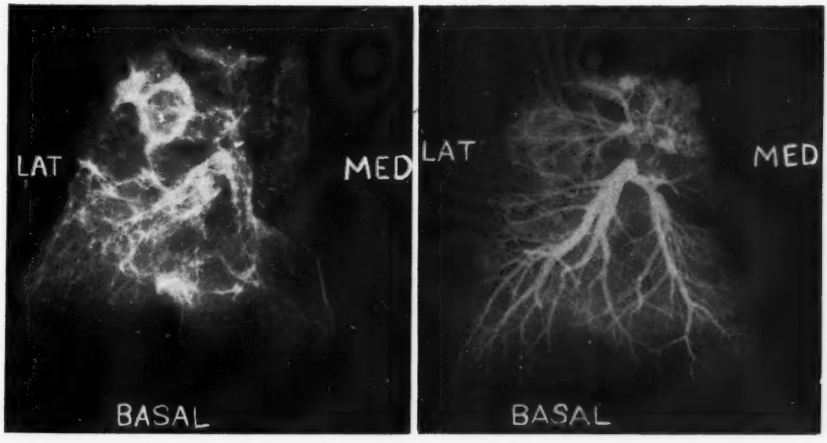
Fig. 8.—Roentgenogram of the injected specimen, taken from the lung shown in roentgenograms in Fig. 7, reveals an overexpanded anterior segment with a relatively normal circulation. The apical segmental circulation is destroyed; since the patient never had hemoptysis, the vessel must have ruptured into the cavity during the course of the injection. The vessels of the posterior segment are thin and straight and show decreased branching. The lingular circulation is relatively abnormal throughout.

not pertinent since there was sufficiently extensive x-ray involvement at the time of surgery to account for the destroyed vascular pattern which was found.



A. *B.*

Fig. 9.—*A*, Roentgenogram shows the height of disease; essentially, the same picture persisted for six months, following which there was gradual subsidence to the picture seen in *B*. *B*, X-ray film taken fourteen months later and immediately prior to surgery. Only residual disease and the cavity in the superior division of the lower lobe are seen. Because of palpable disease in the basal segments, the entire lower lobe was resected. This lower lobe is shown in Fig 10, *A* and *B*.



A. *B.*

Fig. 10.—*A*, This roentgenogram shows an obvious cavity in the superior division, but no striking residual involvement of the basal segments. *B*, Roentgenogram of the injected specimen shows obliteration of the endarterial and precapillary branches of the superior division. but the circulation of the basal segments appears essentially unimpaired. Considering the extent and length of the involvement we are at a loss to explain the excellent status of the circulation.

Fourteen satisfied the original criteria of extensive disease, clearing to relatively insignificant residuals. Four of these cases comprise the present report.

DISCUSSION

It is well accepted that pulmonary tuberculosis causes obliterative endarteritis. There is considerable evidence to suggest that these changes are permanent. Present-day concepts of resection predicate long-term preparation with antibiotics prior to resection of residual caseous foci. It is apparent that any attempt to reconcile these facts reveals a basic discrepancy. If an entire lobe has been involved with disease, arterial impairment of the entire lobe despite marked parenchymal clearing prior to resection might be expected.

Operations designed to remove only the residual caseous foci may eliminate potentially dangerous areas of disease, but they are not necessarily function sparing since poorly vascularized lung remains which is capable of only minimal or negligible gaseous exchange. Conversely, the retention of such nonfunctioning segments does prevent overdistention of the remaining uninvolved lobes, and through a space-filling function it does tend to preserve all available normal parenchyma at optimal volume.

When function testing becomes a practicable procedure for the smaller pulmonary units the way will be prepared for the ultimate solution of this problem.

SUMMARY

1. An attempt has been made to investigate the permanency of endarterial changes in cases of pulmonary tuberculosis which have shown considerable regression under streptomycin therapy.

2. In the main, those segments which were involved with disease will show impairment of their circulation despite later clearing of the parenchymal infiltration. A gross exception to this in a case of lower lobe disease has been presented also.

3. By implication some skepticism is expressed regarding the function-sparing merits of segmental resection in originally extensively involved lobes.

4. The ultimate solution to this problem must await arrival of a satisfactory method for direct function testing of individual pulmonary lobes and their segments.

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EXTRAMUCOUS CLOSURE OF THE BRONCHIAL STUMP WITH CLIPS

An Experimental Study

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PROCEDURES for closing the bronchial stump have been described to such an extent that it can nearly be said that each thoracic surgeon with experience and inventive spirit possesses his own technique. For that reason we omit mentioning the various methods used, already known by all, and we are going to limit ourselves to presenting our original method.

Up to the present time, the procedures employed have made use of various materials (silk, linen, nylon, steel, etc.) which cross the whole bronchial wall and which, fortunately in a very small percentage, produce intrabronchial intolerance signs, with production of granulomas, irritative cough, etc. Such occurrences necessitate the extraction of this material bronchoscopically, or sometimes the patient spontaneously expels it by coughing. This situation made us think that by using special clips which would not cross the mucosa, such complications would be avoided, producing a perfect *extramucous closure* of the stump; it could be considered as a useful method in human surgery, having advantages which those methods described up to the present do not possess; at least, theoretically, it would be more ideal.

MATERIAL AND METHOD

Fifteen mongrel dogs, weighing from 7 to 30 kilograms, were used for this experiment. Left pneumonectomy was done on all except the third dog, in which upper and middle bilobectomy was done.

We used three kinds of clips. The first four dogs were considered a preliminary group from which we could determine if this method would provide airtight closure of the stump and from which we could devise the form and measurements of the clips used in the definitive experiments. On these four dogs we used 12 by 3 mm. skin clips, cutting nearly all their teeth. In the remainder of the animals silver clips as shown in Fig. 1, *A* were used; the measurements were 15 mm. in length, 1.5 mm. in width, and 0.5 mm. thick; we do not think that this is hard enough to be used on human beings. The V2A steel clips (which have 18 per cent chrome and 8 per cent nickel) were 15 mm. in length, 1.5 mm. in width, 1 mm. thick, and had teeth 0.5 mm. in length, which were blunt so that they would not penetrate as shown in Fig. 1, *B* and which, because of their hardness, had to be placed in position with small forceps. These clips, if they were less thick and longer, would probably be the most suitable for human surgery.

This work was carried out at the Chirurgische-Universitätsklinik of Göttingen, Germany. Director: Professor Hellner, M.D.

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Intravenous sodium Evipan was used as the anesthesia; the optimum dose was 50 mg. per kilogram of body weight. After injecting curare in most of the dogs, intratracheal intubation was performed connecting the tube with an automatic respirator; oxygen was administered during the operation and rarely nitrous oxide was used as complement.

The dog was placed in lateral decubitus; we entered the pleural cavity through the fifth left interspace, injecting 1 to 2 c.c. of 1 per cent procaine intrapericardially before the ligature of the pulmonary artery. The pulmonary artery and the veins of the three lobes were dissected and ligated independently; the vagus nerve was dissected and separated so that it would not be injured or crushed with the clips, as occurred with Dog 5.

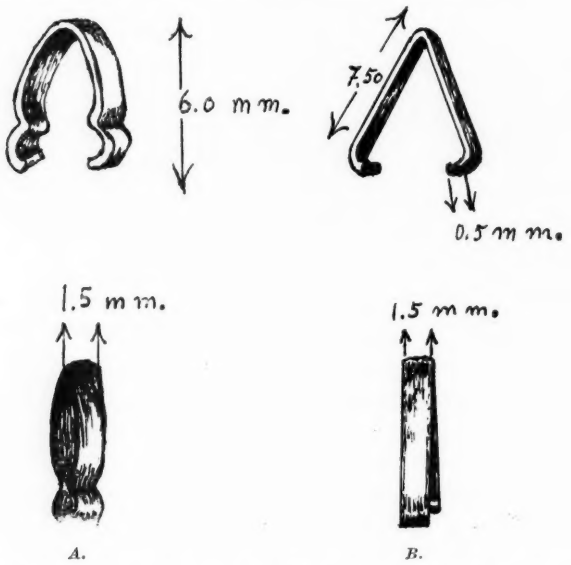
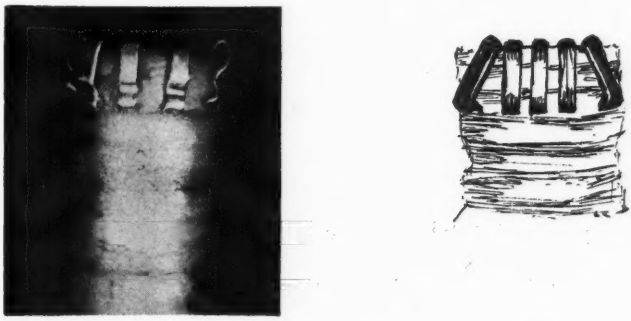


Fig. 1.—*A*, Shape and measurements of the silver clips. *B*, Shape and measurements of the V2A steel clips.

Two stitches of silk were put in opposite points of the bronchial wall to support the stump when the lung had been extirpated, in order to facilitate the placing of the clips. The bronchial stump was eventually closed by a T clamp so that the contralateral lung ventilation would not be disturbed; a clamp was shifted lengthwise, as the clips are placed in position. Then the clips were perpendicularly placed on the edge of the bronchial stump (that is to say, parallel to the longitudinal axis of the bronchial stump), except the two clips at the corners which we placed obliquely either from the outside inward or from the inside outward, as shown in Fig. 2, in order to close the angles completely to avoid the danger which really exists that the clips might slip outward as we observed in some instances when they were placed perpendicularly; we think this detail is important.

After placing the clips (generally four or five were quite sufficient), we acquired a good stump closure; this was proved by the disappearance of the noise produced by the suction of air. Only in Dogs 1 and 3 did we place a free pleural flap obtained from the resected lung, positioning it directly over the stump and on top of the clips. In the rest of the dogs we did not use a pleural flap; neither was the mediastinal tissue sutured nor approximated to the bronchial stump, which was spontaneously buried in its depth. The closure of the chest wall was by layers with catgut; at no time during the operation or the postoperative course did we use sulfonamides or antibiotics. In no case was drainage left nor was the phrenic nerve cut, except in Dog 1. A few days after the operation we obtained radiographs to determine the position of the clips (Fig. 3).



A. *B.*

Fig. 2.—The oblique position of the end clips shown. *A*, Silver clips placed from the outside inward. *B*, Drawing of V2A steel clips placed from the inside outward.

RESULTS

Skin clips were used on Dogs 1, 2, 3, and 4; V2A steel clips were used on Dogs 5 (one skin clip was used) and 8; silver clips were used on Dogs 6, 7, 9, 10, 11, 12, 13, 14, and 15.

The causes of death were: 1. Intentional sacrifice by overdose of sodium Evipan in intravenous injection (the fifteenth postoperative day for Dogs 2 and 7, the thirty-first postoperative day for Dog 11, the one hundred sixty-eighth postoperative day for Dog 13, and the one hundred eighty-eighth postoperative day for Dog 10). 2. Faulty anesthesia (Dogs 1, 4, and 9). These dogs died a few hours after the operation. 3. Abscess of the chest wall (Dogs 8 and 14 on the seventh and thirteenth postoperative days, respectively). 4. Bronchopleural fistula (Dog 5 died on the eleventh postoperative day). 5. Atelectasis of the remaining lung (Dogs 3, 6, and 12 died on the fourth, first, and eleventh postoperative days, respectively). 6. Unknown cause (Dog 15 died the nineteenth postoperative day).

To study the state of the bronchial stump, we had at our disposal ten dogs. In these it was possible to study what occurred in the bronchial stump

and the tolerance of the clips during a period of time varying from four days (Dog 3) to six months (Dog 10).

Dogs 1, 4, 6, and 9 are of no interest because their deaths occurred soon after the operation. The clips in these dogs were still in the positions where they were placed during the operation.

In Dog 5 it was necessary to place a skin clip in the superior end of the stump because of difficulties which arose in the placing of the V2A steel clips due to their hardness. At the necropsy a bronchopleural fistula was observed at the site of the skin clip; the rest of the stump was healed in spite of the fact that a branch of a V2A clip was displaced.

Fig. 3.—Position of the silver clips radiographically observed a few days after the operation. The upper clips are not displaced. Their appearance of displacement is due to the curve of the section of the bronchial stump.

The commentary on Dog 3 is very interesting. Upper and middle bilobectomy was performed and the bronchial stump was closed by placing a free pleural flap, obtained from the upper lobe, over it and on top of the clips immediately. At the autopsy, atelectasis of the lower lobe was observed; about 200 c.c. of sanguineous liquid was present in this pleural cavity; the pleural flap was necrotic and the stump, observed intrabronchially, had a perfect extramucous closure, but without early signs of healing at the edge, which indicates that the necrotic flap hindered the normal healing process.

Dogs 2 and 7, which had no pleural flap or covering of mediastinal tissue, as the others did, were sacrificed the fifteenth postoperative day. The healing of the stump and mediastinal wound was observed and appeared to be completely normal, although in both dogs, two and one clips, respectively, were intrabronchially displaced.

In Dog 8 which died on the seventh day because of an abscess in the chest wall, the healing was good, weak on half of one side, with two displaced clips.

In the rest of the dogs the stump healing was good, with a good extramucous closure, except in Dog 11, in which the teeth of a silver clip formed an intrabronchial prominence. Dog 13, sacrificed the one hundred sixty-eighth postoperative day, and Dog 10, sacrificed six months postoperatively, presented good closure with the clips in good position; there was normal healing, without inflammatory signs of intolerance, which was not microscopically controlled.

COMMENT

The extramucous closure method described complies with the conditions needed to guarantee the obliteration of the bronchial stump; its airtight closure was proved by the disappearance of the noise produced by the suction of air, due to the coaptation of the stump edges. Since the clips are placed parallel to the direction of the vessels there is no interference with the bronchial blood supply; we must have respect for the conservation of the peribronchial tissue which is important in the healing process. There is also no interference with the areolar mediastinal tissue since there is enough space left between the clips. The clips have been well tolerated as demonstrated by the disclosure six months after the operation that a good healing of the stump had occurred, the clips being enclosed in the fibrous tissue, without signs of inflammatory intolerance. The fact must be added that the special characteristic of the method is the *extramucous closure,* that is to say, the clips do not cross the whole bronchial wall but the small teeth cross the peribronchial tissue and penetrate to the cartilage and/or "ligamentum," perhaps to the muscular layer but stay outside the mucosa.

It is possible to object to this method because the quantity of foreign material left in the wound is greater than in other procedures described; above all, as we said happened on various dogs, there is a danger that the clips may be displaced. However, when both edges of the stump were well coapted at the same level and the clips of the corners were placed in a perfect oblique position, so that we were satisfied with the closure obtained, the necropsy showed us the value of this method and confirmed the ideas maintained.

Moreover, the dogs tolerated the experiment and the day after the operation were already walking.

As to which clips are best of the silver or the V2A steel ones, we have to state that with the latter we have had hardly any experience; although we like them, we did not have the desired facilities to obtain them and to make the necessary modifications in their thickness; those used were too hard. Nevertheless, we think, that since they are well tolerated and of greater hardness than the silver ones, they are superior for human beings.

The results obtained without a pleural flap on the stump confirm what Overholt[1] maintained in his book, at least in pneumonectomy.

On the cadaver we have proved the method, obtaining a perfect extramucous closure and with more guarantee than on the dogs since the bronchial wall is thicker.

As the series presented is rather small we think it would be convenient to continue the experiments before deciding to use it on human beings.

SUMMARY

An original method of extramucous closure with special silver and V2A steel clips is described by which a good bronchial stump closure is obtained.

The teeth of the clips do not cross the whole bronchial wall, they respect the mucosa.

The results of using the method in fifteen dogs are discussed, showing the advantages and disadvantages.

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BRAIDED TANTALUM AS A BRONCHIAL SUTURE MATERIAL

Experimental and Clinical Observations

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STAINLESS steel wire is now quite widely used by thoracic surgeons as a bronchial suture material. However, it possesses the disadvantages of being somewhat difficult to handle and of kinking when the knot is being brought down.

An attempt was, therefore, made to find another equally suitable suture material not possessing these disadvantages to the same degree. Such a suture material was found in braided tantalum.

The latter possesses the tensile strength and inertness of stainless steel wire with less tendency to kink. It may be described as being "in between" silk and stainless steel in this regard.

Table I. Results Obtained

DOG	SEX	WEIGHT IN POUNDS	DATE OPERATED	DATE SACRIFICED	OPERATION PERFORMED	TYPE OF SUTURE*	CONDITION OF BRONCHIAL STUMP
1.	M	57	April 2	July 16	Rt. upper lobectomy	CT	Primary healing; stump covered with epithelium
2.	M	24	March 13		Rt. lower lobectomy	IT	No fistula present
			April 1		Rt. upper and middle lobectomy	CT	Bronchus closed and appeared healing well
				April 8	Sucking wound of chest; animal sacrificed		
3.	M	50	April 10	Aug. 14	Rt. pneumonectomy	CT	Stump healed and covered with epithelium
4.	M	54	May 29	July 10	Rt. upper lobectomy	CT	Primary healing end covered with epithelium
5.	F	34	June 4	Aug. 1	Rt. pneumonectomy		This animal was inadvertently sacrificed by another department
6.	M	34	June 12	Oct. 2	Rt. upper and middle lobectomy	IT	Primary healing covered with epithelium
7.	M	26	June 17	Aug. 4	Rt. upper lobectomy	IT	Bronchial stump healed; no fistula
8.	F	38	June 19	July 3	Rt. pneumonectomy	IT	Primary healing; no epithelial covering noted
9.	M	20	June 24	Oct. 2	Rt. upper lobectomy	IT	Primary healing covered with epithelium

*CT — Continuous tantalum.
IT — Interrupted tantalum.

Experimental.—Braided tantalum (0000) mounted on an atraumatic curved needle was used in the experimental work. Nine dogs were subjected to ten operations consisting of various types of pulmonary resection, and 0000 braided

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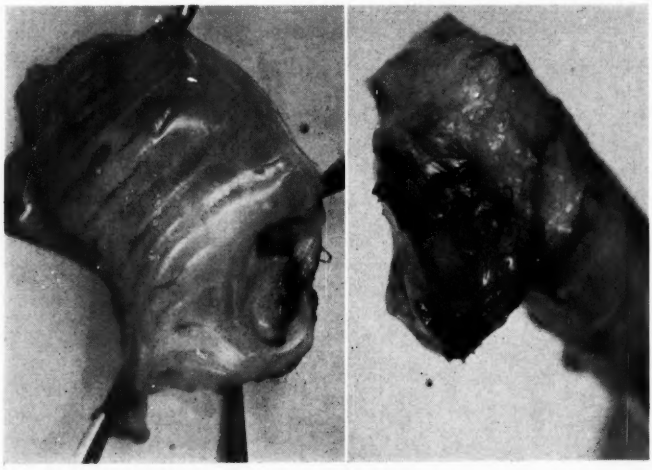
tantalum was used either continuously or in an interrupted manner on the cut end of the bronchus. All nine dogs (with the exception of one which was inadvertently destroyed) were sacrificed after varying periods of time. In no case, at the time of operation, was the bronchial stump covered or an antibiotic used. Table I demonstrates the results obtained.

Examination of the specimens was done by a competent pathologist who found primary healing taking place in all cases. There was no evidence that in these cases healing occurred by opening up of the bronchus and the formation of plug as found by Rienhoff.



A. *B.*

Fig. 1.—*A*, The trachea and bronchial stump inner aspect in Dog 8 fourteen days postoperatively. Note that healing is taking place and the stump has not opened up. *B*, External view of the stump. Sutures are seen. No epithelization is noted as yet at early date.

This suture material was used on four human beings in three lobectomies and one segmental resection without untoward results.

I believe that in human patients a heavier suture such as 000 tantalum would be better to use than 0000 because of the greater thickness and toughness of the human bronchus.

SUMMARY

A new bronchial suture material is described which is equally as good as stainless steel wire but easier to handle. First impressions from a small series of cases indicate that it can be used in tuberculous cases or in other cases where stainless steel wire is used.

The suture material for the experiment was furnished by Johnson & Johnson Co. (Ethicon Suture Laboratories, Incorporated), New Brunswick, N. J.

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PAPILLOMA OF THE BRONCHUS

Case Report

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PAPILLOMAS of the bronchus have been reported, but it is difficult to determine what percentage of benign endobronchial tumors are true papillomas. The literature is not of much help because the name "papilloma" has been somewhat loosely applied to a variety of lesions within the bronchus, some of which are inflammatory in origin and are not true neoplasms.[1] There is no doubt, however, that true papilloma is a rare tumor of the bronchus and an uncommon cause of bronchial obstruction.

A study of four cases filed as papilloma of bronchus in the Department of Surgical Pathology, Toronto General Hospital, revealed that three of them are probably of inflammatory origin. The fourth is probably an endobronchial hamartoma. A recent case which came to our attention, following lobectomy by Dr. F. Kergin, of the Department of Surgery, Toronto General Hospital, more accurately fulfills the requirements of a true papilloma.

CASE REPORT

The patient was a 51-year-old married white woman. She had been in good health, with no symptoms referable to the respiratory system, until three years before admission to this hospital. At that time she had a sudden onset of hemoptysis, persisting for one day, during which she coughed up about one cupful of bright blood. Following this she was perfectly well until one year before admission, when she had a very similar episode. On this occasion chest x-ray pictures were taken, and she was told that she had pneumonia. Bronchoscopic examination was performed and Lipiodol bronchograms were made, without demonstrating any abnormality, and following this she was again well, with no symptoms of any kind.

One month before admission she had another episode of hemoptysis, lasting two to three days, and following this, while she felt well, she developed a persistent cough, productive of a small amount of green sputum. There was no weight loss nor any other symptomatology.

Abnormal physical findings were confined to the right lower chest posterolaterally, where there was diminished air entry and where a few fine râles could be heard. At bronchoscopy a mass of pink tissue was seen in the right lower lobe bronchus, thought to be arising in the superior segmental bronchus to that lobe. A biopsy was reported as normal bronchial mucosa.

At thoracotomy the superior segment of the lower lobe was collapsed. A segmental resection was considered technically impossible, and the right lower lobe was resected. When the lower lobe bronchus was divided a finger-like projection of soft, friable, pink tissue protruded from the opening. When the bronchus was more widely opened the base of the tumor was seen to be attached to the bronchial wall at the junction of the lateral and the anterior basal divisions. Here the mucosa was heaped up in many frondlike projections.

Microscopically, the tumor consisted of papillary mucosal folds, each of which had a core of thin-walled blood vessels and connective tissue (Fig. 1). The epithelium was of a columnar ciliated type, and was two or three layers thick in some areas (Fig. 2). Its

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appearance explained the diagnosis of normal bronchial mucosa reported on the biopsy specimen. The remainder of the lobe showed a chronic interstitial pneumonitis. It was considered unlikely that the tumor was of inflammatory origin, and its histology conformed with the accepted definition of papilloma.

Fig. 1.

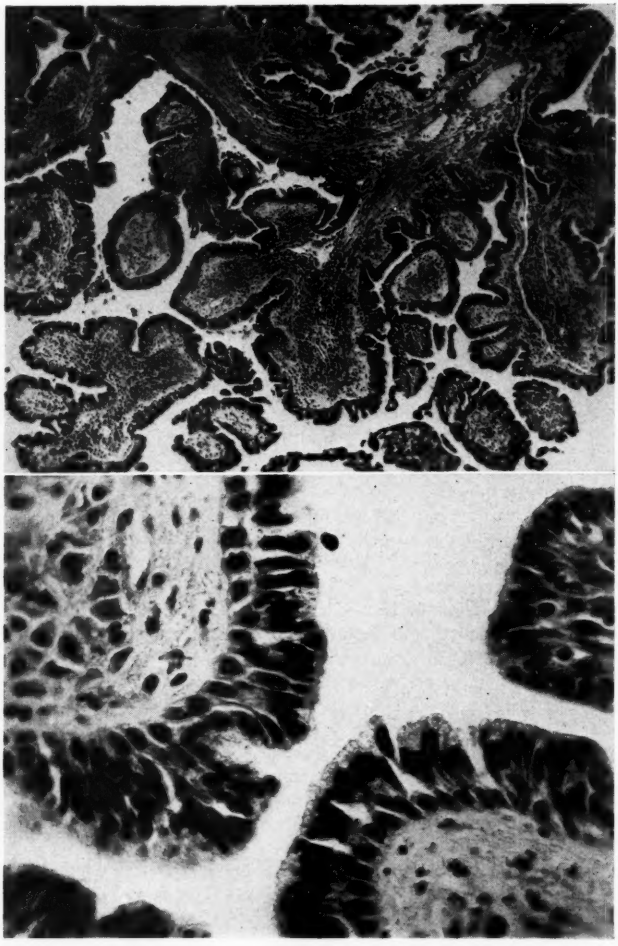

Fig. 2.

Figs. 1 and 2.—Papilloma of bronchus, high- and low-power magnification.

The patient made an excellent recovery, and was discharged from the hospital free of symptoms.

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BRONCHIAL ADENOMA IN CHILDREN

A Case Report of a 7-Year-Old Boy

D. E. Ward, Jr., M.D.,* H. H. Bradshaw, M.D., and Thomas C. Prince, Jr., M.D.
Winston-Salem, N. C.

ADENOMA of the bronchus is a rare tumor in the pediatric age group. Bronchial adenoma was first recognized seventy-one years ago, and yet it received little attention until relatively recently. Only eight cases of adenoma of the bronchus could be found reported in the available literature in children under 14 years of age. These eight cases have been reviewed, and a case of adenoma of the bronchus in a 7-year-old boy treated by pneumonectomy is reported. The recent advances in thoracic surgery and anesthesia have broadened the therapeutic approach to these tumors in children.

Mueller,[1] in 1882, was the first to report a case of bronchial adenoma; he noted it accidentally at the autopsy of a woman who had died from suppurative lung disease which had persisted for eight years. Patterson[2] in 1930 found twenty-six cases recorded in the literature, ten of which were diagnosed at postmortem, and the remaining sixteen were diagnosed during life. Kramer and Som[3] in 1935 recommended bronchoscopic treatment with the use of radon seeds. Chevalier Jackson[4] brought attention to the tumor and encouraged endoscopic removal.

The instance of bronchial adenoma has been stated to be 6 per cent of all primary bronchial tumors. Kramer and Som[3] found 23 adenomas among 353 cases of bronchial tumors. Of 217 histologically proved primary lung tumors encountered at Lahey Clinic since 1930, 15 were bronchial adenomas, an instance of 6.9 per cent.[5] It comprised 12 per cent of 278 neoplasms diagnosed bronchoscopically by Clerf and Bucher.[6] It seems that the tumor is being recognized and diagnosed more frequently as the years go by.

Table I reveals the data on the eight cases of bronchial adenoma found in children under fourteen years of age in the available literature. The patient in the case report included in this article seems to be the youngest in any reported case of bronchial adenoma. The other patients ranged in age from 9 to 13 years. Two patients were treated by pneumonectomy and four were treated by bronchoscopic removal, with a three-and-one-half-year survival reported by Jackson[4] in one patient. No treatment was given for the other two patients.

The following is a case report of a bronchial adenoma in a 7-year-old child treated by pneumonectomy:

CASE REPORT

Chief Complaint.—This 7-year-old white male child was admitted to a hospital Dec. 9, 1952, complaining of "fever."

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Received for publication June 1, 1953.

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TABLE I. BRONCHIAL ADENOMAS REPORTED IN CHILDREN UNDER 14 YEARS OF AGE

AUTHOR		AGE	SEX	LOCATION OF TUMOR	TREATMENT	RESULT
Kramer and Som[3]	1935	13	?	Not reported	Bronchoscopic removal locally followed by radiotherapy	Unknown
Rosenblum and Klein[13]	1935	11	M	Right main bronchus	Bronchoscopic removal locally	Clearing of atelectasis 2 mo. later by x-ray
Peterson[18]	1936	11	?	Unknown	Unknown	Unknown
Jones, MacKenzie, and Biddle[14]	1943	10	M	Right main bronchus	—	Died during bronchoscopy
Jackson, Konzelmann, and Norris[4]	1945	13	M	Right main bronchus	Bronchoscopic removal by forceps and electrocoagulation	Symptomatic cure 3½ yr.
Souders and Kingsley[15]	1948	13	M	Left main bronchus	Partial bronchoscopic removal Left pneumonectomy elsewhere	No follow-up
Som[16]	1949	13	?	Unknown	Bronchoscopic removal	Unknown
Smoller and Maynard[17]	1951	9	F	Right middle lobe bronchus	Pneumonectomy with thoracoplasty	Excellent—10 mo. P.O.

Present Illness.—The child had been in apparent good health until June, 1952, at which time he developed "pneumonia" of the left lung, which was treated with penicillin. Subsequently, he developed a cough which persisted for several weeks and he complained of a pain in the left thorax. Although he improved, he was never well according to his parents.

In October, 1952, he again developed fever, and the condition was diagnosed as "pneumonia" of the left lung. He was treated with penicillin with improvement. On Nov. 1, 1952, a chest x-ray picture revealed a density suggesting an atelectasis of the left lower lobe. The patient was treated with Chloromycetin and sulfa drugs. Improvement was again noted, but on Nov. 28, 1952, the patient had a high fever and was returned to the hospital. A chest x-ray film showed atelectasis of the left lower lobe, and the patient's temperature was 103° F. The child was treated with penicillin and Terramycin, but failed to improve. A diagnosis of bronchial obstruction was made and the child was transferred to the North Carolina Baptist Hospital, Winston-Salem, N. C., for further treatment.

Physical Examination.—On admission the temperature was 104° F., pulse 120 per minute, respiration 28 per minute, and blood pressure 96 systolic over 60 diastolic. He was a thin, pale-looking boy, who appeared chronically ill and weighed 41 pounds.

The trachea and heart were found deviated to the left side. The chest was asymmetrical with increased expansion over the right side. Tactile fremitus was absent over the left side except over the upper lobe. Dullness to percussion was present over the left lower chest, and breath sounds were absent over the left lower lobe. No râles were heard. No murmurs were heard in the heart, and no organs or masses were palpable in the abdomen. No lymph nodes were enlarged or tender.

Laboratory Data.—Hemoglobin was 14 Gm., red blood cell count 4,600,000, corrected sedimentation rate 48 mm. per hour, white blood count was 26,750, with 80 per cent polymorphonuclearphils and 20 per cent lymphocytes. Urinalysis was negative. Wassermann and Kahn tests were negative. Alpha streptococcus and nonhemolytic staphylococcus were cultured from the throat. Three blood cultures produced no growth. Tuberculin skin test was negative. Chest x-ray film (Fig. 1) showed evidence of atelectasis of the left lower and left upper lobes with marked shift of the mediastinal structures toward the left side. No foreign body was visualized.

Course in the Hospital.—On Dec. 11, 1952, at bronchoscopy a tumor in the left main bronchus (Fig. 2) was visualized. A biopsy was taken which the pathologist thought represented a bronchial adenoma. On Dec. 15, 1952, a left pneumonectomy was done under endo-

tracheal Vinethene, ether, and oxygen anesthesia. The left lower lobe was found to be atelectatic, firm, and hard. The left upper lobe was emphysematous and would not deflate.

A left pneumonectomy was done by individually ligating the arteries and veins. The left main-stem bronchus was opened and a sessile firm lesion attached to the superior wall of the bronchus was found, measuring 0.7 by 1.0 by 0.5 cm. in size. The left main bronchus

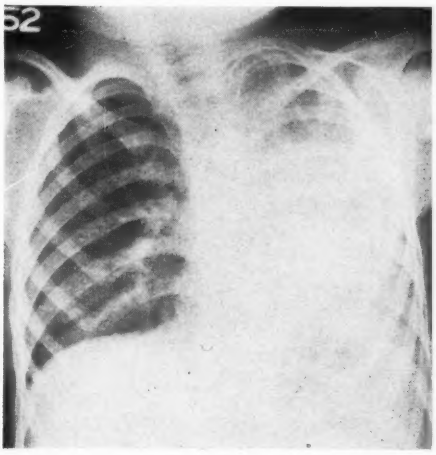

Fig. 1.—Preoperative chest x-ray film showing atelectasis of the left lung.

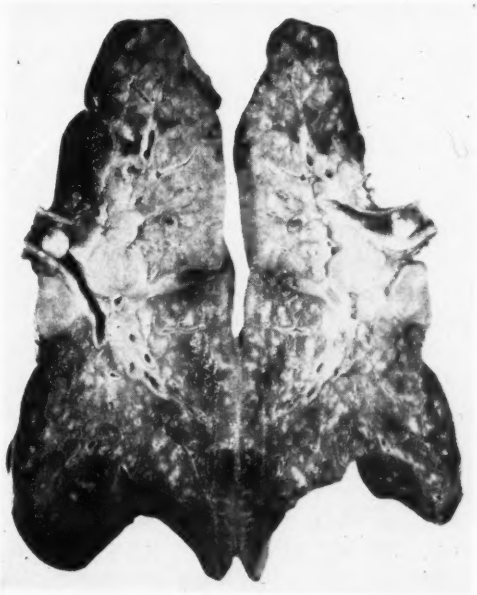

Fig. 2.—Bronchial adenoma in left main bronchus with extension into the lung, but not into the lymph nodes.

was severed proximal to the tumor and was closed with interrupted sutures. A frozen section obtained on a hilar lymph node during the operation was reported negative for tumor. A partial thoracoplasty was done with resection of the fifth, sixth, and seventh ribs on the left side.

Postoperative Course.—The patient was given daily penicillin and dihydrostreptomycin and had an uneventful postoperative course. Fig. 3 shows the postoperative x-ray picture. The child was discharged on the ninth postoperative day in good condition. Three months later the child was examined and had no cough, chest pain, or other respiratory symptoms.

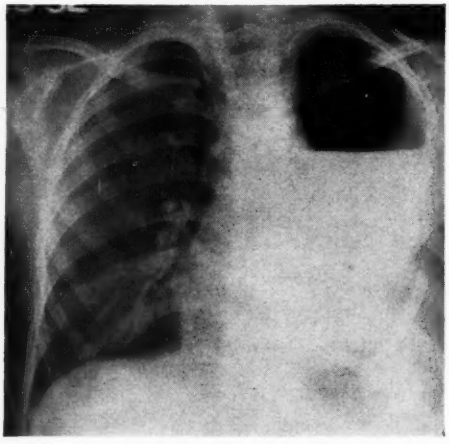

Fig. 3.—Postoperative chest x-ray film taken after left pneumonectomy.

DISCUSSION

Bronchial adenoma is an uncommon tumor below the age of 14 years. A review of the literature shows that the lesion does occur in children and should be thought of when bronchial obstruction is found. The symptomatology of bronchial adenoma is that of any other type of bronchial obstruction. An important symptom of bronchial adenoma is hemoptysis, which is attributable to the marked vascularity of the tumor. A definite diagnosis can be established by bronchoscopy and biopsy of the lesion.

Bronchial adenoma may be locally invasive or may metastasize by way of the lymphatics or blood stream.[7, 8] Metastases to the liver, vertebral bodies, opposite lung, and kidney have been reported.[7-10] Goldman[11] presented a report on five patients showing malignant qualities in bronchial adenomas. Clerf and Bucher[6] reported a case of bronchial adenoma in which biopsy from the bronchus was examined histologically at intervals over a five-year period, at the end of which time carcinomatous change was detected in the tumor. That finding was confirmed at autopsy. Umiker and Storey[12] reported a case of peripherally located bronchial adenoma with a histologic evidence of transformation into adenocarcinoma six years following detection. A right upper lobectomy was done and the tumor was reported as "well differentiated adenocarcinoma."

There has been much discussion in the past concerning the treatment of adenoma of the bronchus. It is never possible to tell from bronchoscopic examination whether a tumor extends through the bronchial wall as a "dumbbell" type of tumor or into the surrounding lymph nodes. It has been proved that bronchial adenoma may grow and metastasize as a tumor of low-grade malignancy. It seems clear now that excision of the entire tumor and surrounding tissue is the only adequate treatment. This can best be done by a lobectomy or pneumonectomy.

SUMMARY

A case of bronchial adenoma in a 7-year-old boy treated by pneumonectomy is presented. A review of the literature of eight cases of bronchial adenoma reported under fourteen years of age is included. A plea is made to treat bronchial adenoma as a malignant lesion with pneumonectomy or lobectomy as the only treatment available at present to cure the tumor.

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ENDOBRONCHIAL HAMARTOMA

Report of Two Cases

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THE TERM hamartoma was first introduced in 1904 by Albrecht,[1] who used it in reference to benign mixed tumors occurring in various organs. He described them as tumor-like malformations due to abnormal mixing or abnormal development of the normal tissue components of the organ in which they occur. This abnormality may be the result of variations in the quality, arrangement, or degree of differentiation of the tissues. Many lung hamartomas have been reported as chondromas, lipomas, osteochondromas, etc., but are hamartomas according to Albrecht's criteria. Yet, pure lipomas[2] and fibromas[3] have been reported. Hamartomas are generally described as circumscribed or diffuse, the former being more common. Most often they occur in the periphery of the lung and produce few, if any, symptoms. They are said to occur about once in each 400 individuals[4] and are more common in male patients.

This article, however, deals only with the endobronchial hamartomas which are much less common and which frequently produce symptoms because of obstruction of the bronchi. The usual symptoms are cough, hemoptysis, purulent sputum, wheezing, pain, fever, and dyspnea, in that order of frequency.[5] Recently we have observed two of these unusual endobronchial tumors.

CASE REPORTS

Case 1.—J. N., a 59-year-old white man, was hospitalized Jan. 12, 1953, with a three months' history of a cough productive of whitish, mucoid sputum, occasionally streaked with blood. He denied dyspnea, chills, fever, and weight loss. His physician told him that chest x-ray examination disclosed an "opacity of the lung." He was transferred to this hospital Feb. 11, 1953, for definitive diagnosis and treatment.

The physical examination was essentially negative, and the routine laboratory examinations were not remarkable. The chest x-ray examination, however, disclosed a mottled infiltration in the lower right lung field, felt to be pneumonitis.

Bronchoscopy showed a bluish-red knobby tumor at a division of the right middle lobe bronchus. A previous bronchoscopy at another Veterans Administration hospital had also disclosed this polypoid tumor, but the biopsy was not diagnostic. The biopsy taken here showed fragments of respiratory epithelium, fat, hyaline cartilage, and bone; hamartoma was considered the likely diagnosis.

On Feb. 17, 1953, a right middle lobectomy was performed, and the patient had an uneventful postoperative course.

Pathology.—The gross specimen (Fig. 1) was a firm, atelectatic right middle lobe. At the bifurcation of the middle lobe bronchus was a firm, pink, bosselated tumor mass measuring approximately 3.0 cm. in length and 1.0 cm. in diameter. This arose from the wall of a tertiary bronchus near its origin.

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Fig. 1.

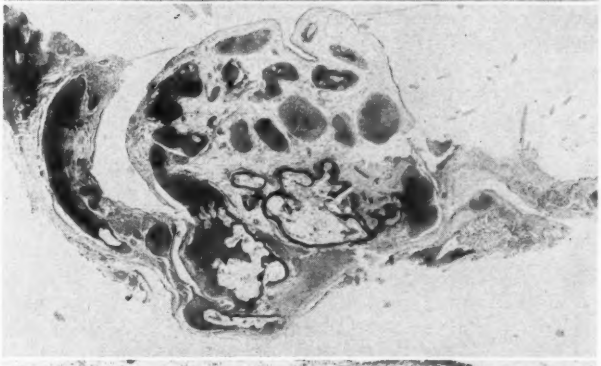

Fig. 2.

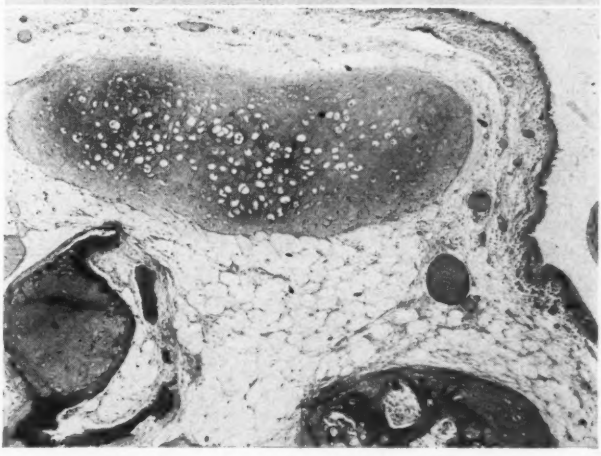

Fig. 3.

Fig. 1 (Case 1).—Note tumor arising near bronchial bifurcation and atelectatic lung tissue.

Fig. 2 (Case 1).—Cross section through opened bronchus showing relation of tumor to bronchial wall. Note cartilage masses, many with metaplastic bone and marrow spaces.

Fig. 3 (Case 1).—Higher magnification of Fig. 2.

Microscopically, the tumor consisted of cartilage masses in disorganized arrangement with fat and myxoid connective tissue between. Many of the pieces of cartilage were undergoing ossification with formation of thin trabeculae of bone and fat-filled marrow spaces, and a few of the marrow spaces contained marrow elements (Fig. 3). The largest mass of cartilage and bone had its origin from cartilage in the deeper part of the bronchial wall (Fig. 2). The tumor surface was covered by respiratory epithelium, which in a few areas showed squamous metaplasia.

Sections from the lung parenchyma disclosed a cellular fibrous pleura infiltrated with chronic inflammatory cells. The alveolar walls were thickened and fibrous, the bronchi were moderately dilated, and a few contained purulent exudate. The alveolar spaces showed hemosiderin-laden macrophages in moderate numbers.

CASE 2.—R. W., a 62-year-old white man, was admitted to a distant hospital on Jan. 30, 1953, following an episode of coughing and hemoptysis. There were no such previous episodes, and the past history was noncontributory. There had been no fever, pain, or chills. On Feb. 18, 1953, he was transferred to this hospital for further study and treatment.

Physical examination was essentially negative. Sputum was smeared and cultured for acid-fast bacilli and fungi and reported negative. Serial x-ray films disclosed emphysema of the right lower lobe with a suggestion of a right hilar density which possibly represented a tumor in that area obstructing the right lower lobe bronchus.

On Feb. 25, 1953, a bronchoscopy disclosed a pink, globular tumor arising from the posterolateral wall of the right intermediate bronchus. This was biopsied, but only respiratory epithelium showing some squamous metaplasia was obtained. A previous bronchoscopy at another Veterans Administration hospital had also shown this tumor.

On March 9, 1953, a right middle and lower lobectomy was performed. The postoperative course was uneventful.

Pathology.—The gross specimen showed near the line of resection of the bronchus, a polypoid, fusiform mass measuring 3.0 cm. in length and 1.2 cm. in diameter (Fig. 4). It was smooth and pink, soft in some areas, and firm in others. On section it contained calcified cartilage (Fig. 5) and arose from the wall of one of the branches of the middle lobe bronchus. The bronchi were reddened and contained much mucoid material. The middle lobe was completely atelectatic; the lower lobe was crepitant.

Microscopically, the tumor was covered with respiratory epithelium showing areas of squamous metaplasia. The stroma was composed of a well-vascularized myxoid fibroreticular tissue. There were fat cells, mucus glands, and masses of cartilage present. Numerous bundles of smooth muscle were found beneath the epithelium but not within the cartilage. There were small inclusions of fat cells within the cartilage. Most of the tumor was composed of an irregular lobulated mass of cartilage, but multiple sections disclosed other smaller separate collections of cartilage cells in the base of the tumor. In many areas the cartilage showed degenerative changes, but no osseous metaplasia was present. The lung parenchyma showed areas of emphysema, foci of round cells, and some diffuse scarring of the alveolar walls. There was atelectasis and fibrosis present in the middle lobe.

DISCUSSION

A review of the literature discloses twenty-four cases of endobronchial hamartoma reported. Postlethwait, Hagerty, and Trent[6] in 1948 reviewed twelve cases and reported one of their own. In 1951 Effler and Scheid[7] added another case. Sherrick[5] in 1952 collected nineteen cases and added still another. Rubin and Berkman[8] reported twenty-eight cases where hamartomas were found at autopsy, and one of these arose from the right middle lobe bronchus. They also included three other cases where pulmonary resections had disclosed

hamartomas, but none of these were endobronchial. Recently, Nissen and his associates[9] reported a case of osteochondroma of the bronchus, making a total of twenty-four, including our own cases.

Of the twenty-four cases now reported, thirteen arose in the right lung and eleven in the left lung. Seven were incidental findings at autopsy, and the other seventeen produced symptoms.

Womack and Graham[10] felt that these tumors represent failures of embryonic bronchial buds to develop normally. Other etiological possibilities

Fig. 4.

Fig. 5.

Fig. 4 (Case 2).—Smooth ovoid tumor blocking bronchus to right middle lobe. Note atelectatic lung tissue.

Fig. 5 (Case 2.)—Microscopic picture showing cartilage with areas of degeneration.

mentioned are that these growths represent hyperplasia of normal structures, reactions to inflammation, and true neoplasms. The most generally accepted belief, however, is that they are congenital in origin.

Most hamartomas show a preponderance of hyaline cartilage, but a careful search of sections from the tumors often discloses, in addition, fat, bone, and epithelial derivatives. The cartilage often undergoes calcification and sometimes even ossification as in our Case 1, where fat-filled marrow spaces were present. Marrow elements may be present as was noted by Nissen and his co-workers[9] and in our Case 1. Marrow elements are occasionally found in ectopic bone formation so probably do not constitute a true part of the tumor. Fat in most of these tumors is probably only a filler material representing metaplastic change in connective tissue. Fat has been noted to occur normally in the bronchial wall.[2] The muscle likewise probably represents only a normal component of bronchial wall, and the epithelium merely covers the tumorous projection or lines passages through the growth.

The presence of bone with the cartilage suggests the diagnosis of osteochondroma, but we feel that the bone merely represents a metaplastic change with aging of the hyaline cartilage. Ossification of bronchial cartilage rings has been noted in adults.[2] Thus the principal component of these tumors is cartilage. In most cases where the type of cartilage has been identified, it has been of the hyaline type, as was true in our two cases. Moore,[11] however, found elastic cartilage in his case and postulated that the cartilage was ectopically placed.

Liebow[4] has separated the chondromas and osteochondromas from the hamartomas, but a review of the microscopic features of the reported endobronchial hamartomas shows no clear-cut features for differentiation of these groups. For the purposes of simplification it seems desirable to include these tumors all in one group, the hamartomas, since this term is inclusive and deeply entrenched in medical literature.

These tumors, particularly those situated in the lung periphery, are now diagnosed more frequently with the increase in pulmonary resections for silent lung lesions. Though these are almost always benign, the true nature of the tumor is disclosed only by operation and microscopic study of the tumor.

Endobronchial hamartomas have occasionally been removed through the bronchoscope, and none have been reported to recur. In other instances, as in our own cases, the tumors are not favorably situated for this type of procedure. Often they produce recurrent bouts of pneumonitis and atelectasis and make it desirable to remove the involved segment. The most frequent symptoms noted have been cough and hemoptysis. The roentgenograms in our cases were not diagnostic of bronchial tumor.

SUMMARY

Two cases of endobronchial hamartoma are reported and discussed. Both were treated by lobectomy.

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A PRACTICAL PHYSICAL METHOD FOR THE DETECTION OF EARLY RESPIRATORY ACIDOSIS DURING THORACIC SURGERY

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PREVIOUS work from this and other laboratories has adequately demonstrated the common occurrence of acidosis during thoracic surgery.[1-7] We have presented evidence that such acidosis stems from inadequate alveolar ventilation during the period of anesthesia.[1] Similar conclusions were reached by Taylor and Roos[3] in 1950 and by von Bühlmann and Hotz[7] in 1951. The previous studies from this laboratory were performed during open-chest procedures while the patient was anesthetized with intravenous Pentothal Sodium-curare. Ventilation was accomplished by intermittent positive pressure (manual) through an endotracheal tube.

This article is a description of a simple method for the detection of an early and slight rise in alveolar PCO_2.

METHODS

Many of the methods used in this work were the same as those used in the determination of alveolar ventilation during thoracic surgery.[1] In addition, pressure tracings were recorded by means of a strain gauge from one of two points: (1) the trachea by means of a polyethylene tube (polyethylene, size 240) inserted through the endotracheal tube, and (2) at the anesthesia bag (Fig. 1). In several instances recordings were taken from both points simultaneously and recorded on a single tracing by means of a two-channel Sanborn recorder. The PCO_2 threshold of the respiratory center was checked by frequent determinations of the arterial blood PCO_2 in the manner previously described. An indwelling polyethylene tube (polyethylene, size 90) in the brachial artery gave ready access to arterial blood samples.

Such pressure recordings were made in fourteen patients who were undergoing thoracic surgery for tuberculosis and who were under Pentothal-curare anesthesia with intermittent manual positive pressure ventilation (controlled ventilation). Eighty-four determinations were made of arterial blood PCO_2 in order to establish the respiratory threshold for PCO_2 under the conditions of an open-chest and Pentothal-curare anesthesia.

The pressure tracings taken from the trachea and the anesthesia bag varied both quantitatively and qualitatively (Fig. 2*A*). The differences were accounted for primarily by the lag in the system created by the resistance in the tubing from the anesthesia bag to the end of the endotracheal tube. The patient's spontaneous respiratory efforts created greater negative pressure in the trachea than in the anesthesia bag (Fig. 2*B*). Since it was the detection of the patient's own respiratory effort that concerned us, the endotracheal tracing proved to be the more sensitive for this study.

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RESULTS

A high degree of correlation was found between the level of the arterial blood PCO_2 and the presence or absence of spontaneous respiratory efforts by the anesthetized patient (Pentothal-curare) with an open thorax. In the presence of an elevated PCO_2 the patient made spontaneous respiratory efforts whenever there was a pause in the intermittent positive pressure administered by the anesthesiologist. In the presence of a normal or low PCO_2 in the arterial blood there were no such spontaneous respiratory efforts on the part of the patient. The pressure tracing was flat during pauses of 5 to 10 seconds in the intermittent positive pressure when the PCO_2 was normal or low. This indicated the presence of true apnea at these times. Such pauses were marked by rhythmic negative pressure fluctuations when the PCO_2 was elevated (Figs. 2 through 6). Pauses in intermittent positive pressure administration are denoted by the word "test" in Figs. 2 through 6.

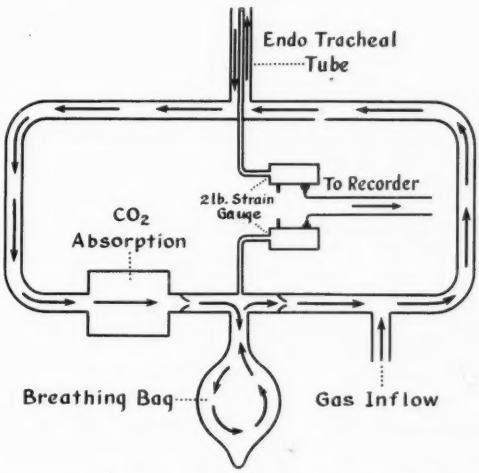

Fig. 1.—Diagram of the anesthesia machine showing the positions from which pressures were recorded. The polyethylene tube was introduced into the endotracheal tube through a tight-fitting hole in the screw-cap of the elbow adaptor.

In order to check the dependability of this relationship eightyfour determinations of arterial blood PCO_2 were made. Each PCO_2 determination was made in direct temporal conjunction with a pause by the anesthesiologist. In the fourteen patients the highest PCO_2 found when the endotracheal pressure tracing indicated apnea was 55 mm. Hg. The one patient who showed the significantly elevated respiratory center threshold was deeply anesthetized according to the anesthesiologist in charge of the case. In all other instances the endotracheal pressure tracing showed spontaneous respiratory efforts whenever the PCO_2 reached 45 to 47 mm. Hg.

As a further test of the validity of the method one patient was purposely anesthetized quite deeply. The PCO_2 and pressure during a pause in the inter-

mittent positive pressure were then determined. This patient had shown a good correlation previously with a threshold of PCO_2 at about 45 to 47 mm. Hg during the first ninety minutes of the intrathoracic procedure. When he was quite deeply anesthetized by a booster injection of Pentothal-curare intravenously, a PCO_2 of 80 mm. Hg in the arterial blood elicited no spontaneous efforts. Whether this lack of spontaneous effort was due to paralysis by curare or to dulling of the respiratory center by Pentothal could not be determined.

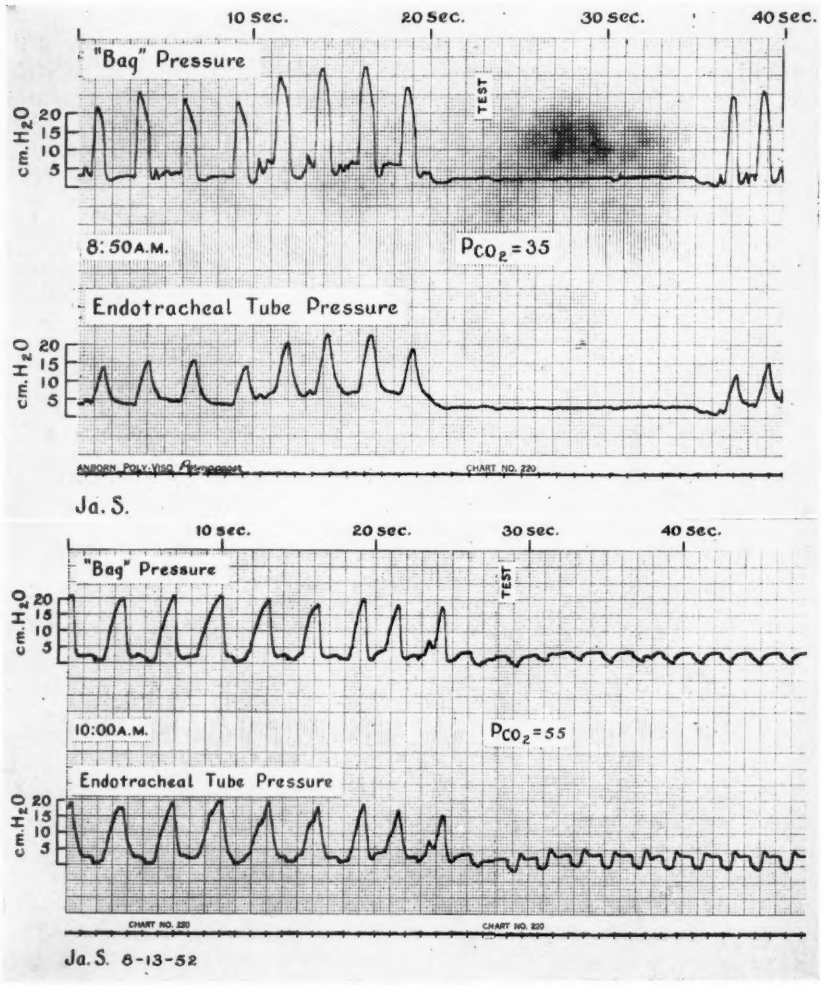

Fig. 2.—*A*, Simultaneous tracings from the endotracheal tube (below) and from the anesthesia bag (above). The area marked "test" represents a twenty second lapse in the intermittent positive pressure and the absence of spontaneous respiratory efforts indicate true apnea at a time when the PCO_2 was below the threshold. *B*, The same patient at a later time when the PCO_2 exceeded the threshold of the respiratory center. During the "test" period spontaneous respiratory efforts are clearly seen. The negative pressures are more clearly shown in the tracing from the catheter in the endotracheal tube.

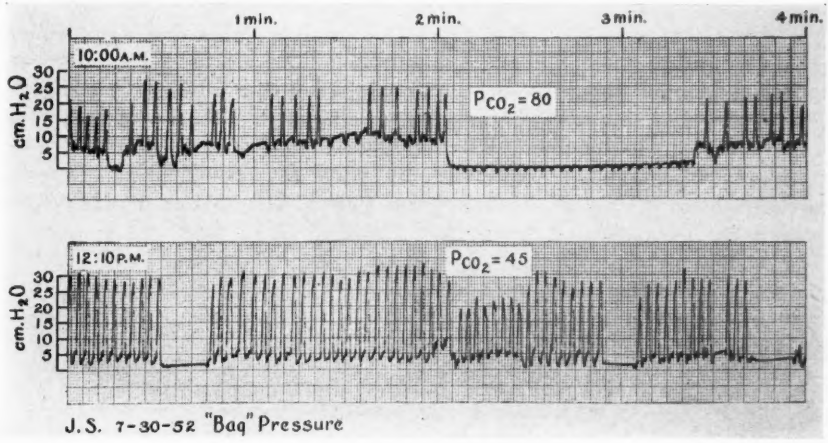

Fig. 3.—Spontaneous respiratory efforts were present at a PCO_2 of 80 mm. Hg and proved to be absent later when the PCO_2 had been brought down to 45 mm. by more vigorous ventilation.

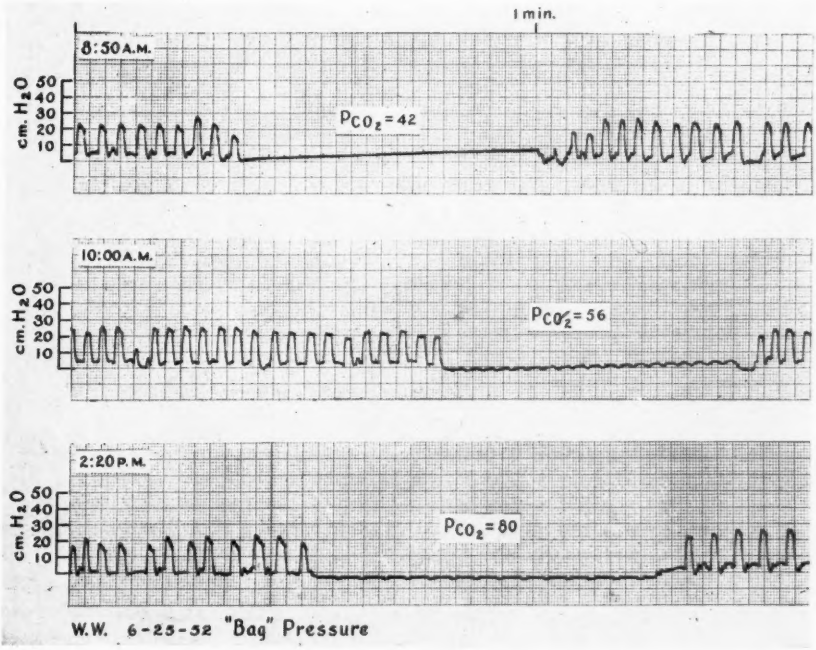

Fig. 4.—Results obtained with patient W. W. during the first operation. Apnea existed during a pause in intermittent positive pressure when the PCO_2 was 42 mm. Hg. Spontaneous respiratory efforts were seen at a PCO_2 of 58 mm. and also later at a PCO_2 of 80 mm. These pressures were recorded from the anesthesia bag. See Fig. 7 for the findings during a second operation on this same patient.

DISCUSSION

Some comment is in order concerning the value of maintaining a near-normal physiologic state during major surgery. The patients whose PCO_2 was maintained near normal showed a more rapid recovery from the anesthesia than did those whose PCO_2 was elevated significantly. It is common for a patient anesthetized with Pentothal-curare not to require much drug during the last hour of a major procedure. It seems likely that in such instances rather severe degrees of CO_2 accumulation may play a definite role in maintaining anesthesia and in slowing recovery.

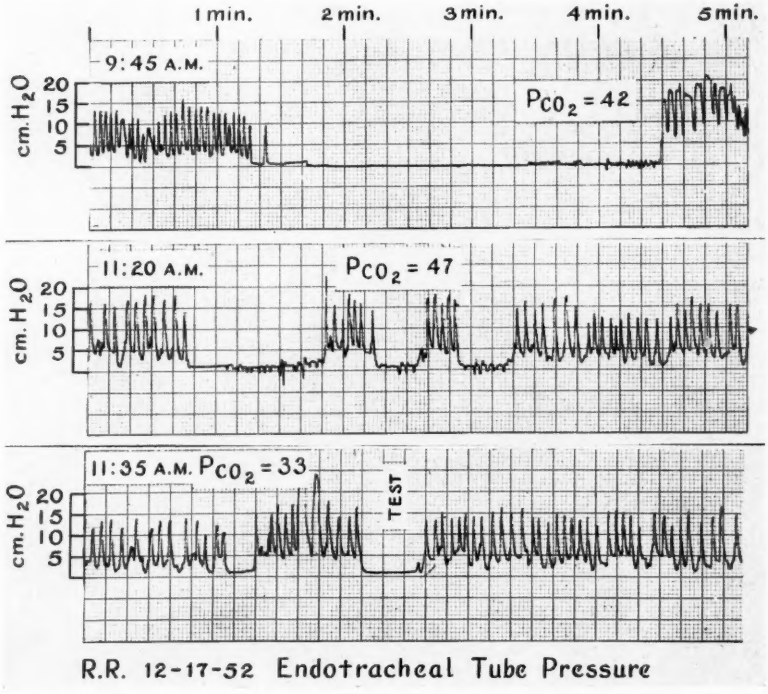

Fig. 5.—Endotracheal tube pressure recording shows the early development of spontaneous respiratory efforts at a PCO_2 of 42 mm. Hg. Later at a PCO_2 of 47 mm. the magnitude of these efforts was better developed. At a PCO_2 of 33, true apnea existed during pauses in artificial ventilation.

Tachycardia is not uncommon during major thoracic surgery. In this series of patients acidosis was frequently accompanied by tachycardia. This would suggest a toxic effect of acidosis upon the myocardium and might explain some cases of cardiac arrest or ventricular fibrillation during anesthesia. Acidosis is particularly dangerous in the presence of anoxia because the two abnormalities are probably additive in their detrimental effect upon the body. We have observed such a combination in one patient who suffered an irreversible cardiac arrest. This event had been preceded by a two-hour period of tachycardia.

The pressure exerted upon the bag varied greatly from one anesthesiologist to another and even during a given procedure under one anesthesiologist. The pressure range was about 15 to 35 cm. water positive pressure. In general, after a long pause (such as for the starting of a transfusion), there was a tendency to give rapid respirations of high pressure (25 to 35 cm. water) and to hold the bag between compressions so that there was an expiratory resistance of 6 to 10 cm. water. Such respiratory assistance is obviously not as efficient as a slower rate and more complete emptying of the lung between respirations.

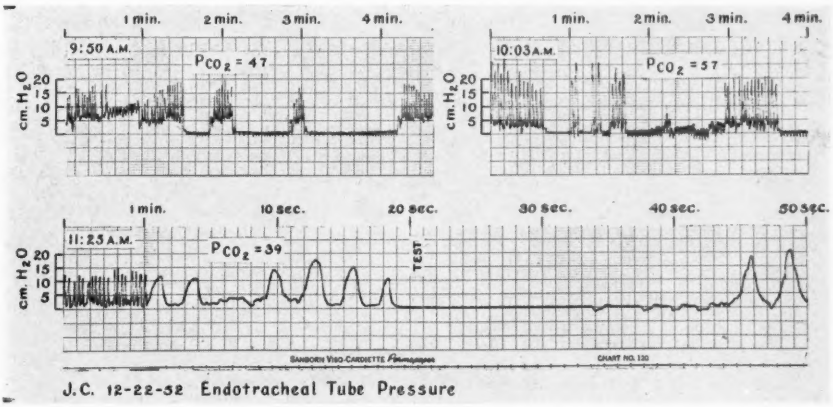

Fig. 6.—Spontaneous respiratory efforts were present at PCO_2 levels of 47 and 57 mm. Hg. The lower tracing shows no such efforts in the early part of the test period when the PCO_2 was 39 mm. Hg. After fifteen seconds, however, such spontaneous efforts appeared, at a time when the PCO_2 crossed the threshold of the respiratory center. This is the ideal PCO_2 level at which to carry the patient.

It was found that it was easier to maintain adequate ventilation by deliberate pressure of moderate magnitude (15 to 20 cm. water) at a rate of 12 to 14 per minute with complete release of pressure between bag compressions. The lack of expiratory resistance makes complete expiration easier and thus makes for a greater exchange of air. The expiratory phase should be at least twice as long as the inspiratory (positive pressure) phase in order to allow for complete emptying of the lungs. During the course of surgery on one lung some positive pressure during expiration is desirable to prevent complete collapse of the operated lung. But at times when deep ventilation is needed to eliminate CO_2 the expiratory resistance should be minimal.

Maloney, Derrick, and Whittenberger[8] have reported on the use of the Emerson anesthesia assistor during ether anesthesia. They report an easier anesthesia with little restlessness, and a quiet mediastinum, indicating adequate ventilation and the absence of respiratory acidosis. The initial purpose of the present study was to determine whether such a demand assistor would be applicable to the patient under Pentothal-curare anesthesia and with an open thorax. The recordings shown in Figs. 2 through 6 demonstrate that under ordinary conditions patients under this type of anesthesia have active respiratory centers and are capable of creating a negative pressure of 1 to 3 cm. water within the trachea. Under heavy anesthesia either the respiratory threshold or muscular strength

may fail, but this is probably uncommon in usual practice. Whether such negative pressures will be adequate to activate the Emerson assistor remains to be seen.

Maloney, Derrick, and Whittenberger[8] have pointed out that good relaxation is easier to obtain in the presence of good ventilation than when the patient is restless due to acidosis. Actual struggling was noted in some of our patients when the PCO_2 was moderately elevated (PCO_2 of 55 to 70 mm. Hg) in spite of the actions of the Pentothal and curare. Probably in some instances more anesthetic agent is given in order to abolish the restlessness which is due to acidosis. More adequate ventilation might control the restlessness without the use of more anesthetic agent. Certainly the respiratory center was active in

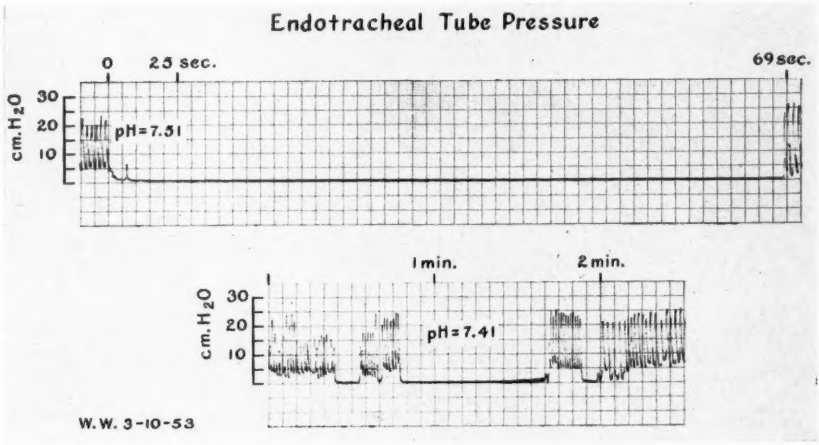

Fig. 7.—By the use of the endotracheal pressure tracing this patient (Patient W. W., second operation) was actually maintained in mild respiratory alkalosis throughout this second procedure (pH 7.51). True apnea can be seen in the pauses. At the latter part of the pause in the lower segment of the tracing respiratory efforts were seen to develop. These negative pressure fluctuations appeared about thirty seconds after the pH had been found to be 7.41. After a few good ventilations true apnea was again observed during a brief pause in artificial ventilation.

the patients whose ventilation was controlled in accordance with the prsence or absence of apnea during pauses in the intermittent positive pressure. Anesthesia was quite adequate in these subjects without the necessity of deepening the level of anesthesia beyond the usual range.

The practical application of such a method of detection of early respiratory acidosis is obvious. It enables the anesthesiologist to observe the pressure pattern throughout the surgical period and to maintain a more nearly physiologic PCO_2. This was actually done in the last nine patients in this study by means of the direct writing recorder. Fig. 7 shows the tracing of a second thoracotomy in patient W. W., whose first operation is represented in Fig. 4. By observing the recording of the endotracheal pressure the anesthesiologist was able to maintain a mild respiratory alkalosis during the second operation.

It is felt that during endotracheal intermittent positive pressure respiration some form of endotracheal pressure tracing is of great value. It would constitute a valuable tool in teaching proper technique of intermittent positive pressure respiration. The form of pressure curve clearly shows the manner in which each pressure is applied, the magnitude of the pressure reaching the trachea, the length of inspiration and of expiration, the resistance to expiration, and the rate of respiration.

It is quite likely that the more experienced anesthesiologist actually feels with his sensitive fingers the same pressure fluctuations which we have recorded and thus knows when to increase his efforts. Nevertheless, reports from various parts of the country have shown that acidosis during intrathoracic surgery is the rule rather than the exception. For this reason it is felt that any aid in detecting the development of acidosis should be useful. As yet there are no satisfactory and simple means for the continuous recording of the pH, PCO_2 or CO_2 content directly. The method described in this paper gives a simple indirect means of following the PCO_2 during thoracic surgery under ordinary anesthetic level. It is now planned to substitute an ordinary sensitive aneroid manometer or an oscilloscope for the recorder for the detection of spontaneous respiratory efforts. A report on a simplification of the present method will be made subsequently. It is logical that some such simple means should be practical.

Finally it should be emphasized that no method for the detection of acidosis can substitute for good clinical judgment and experience in anesthesiology. The method described in this paper is offered only as an aid to the anesthesiologist in his difficult and all-important work.

SUMMARY

1. A simple physical recording of endotracheal pressure during positive pressure has been described.

2. The respiratory threshold for PCO_2 is only slightly elevated and muscular strength is sufficient under the usual Pentothal-curare anesthesia even during operations involving an open thorax to permit the creation of detectable negative pressure fluctuations in the trachea.

3. Thus, the presence or absence of true apnea gives an accurate indication of the absence or presence of a significant respiratory acidosis. By the use of this method any serious degree of CO_2 retention can easily be avoided.

4. The maintenance of a near-normal physiologic state with respect to acid-base balance is probably quite important in the prevention of cardiac arrest and in promoting prompt recovery from the anesthesia.

5. A suggestion is made for a further improvement in the technique to make it more easily applicable in the operating room. This consists of using a bright-line oscilloscope or a sensitive aneroid manometer instead of a direct writing recorder for the detection of spontaneous respiratory efforts.

6. Ancillary values of such a pressure tracing in teaching of anesthesia technique have been discussed.

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PREGNANCY AND TUBERCULOSIS

A Report of Ten Cases Treated by Major Thoracic Surgery

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AT THE Nova Scotia Sanatorium during the past fifteen years major thoracic surgery has been performed to control tuberculosis in ten pregnant patients. In one-half of them, thoracoplasty operations were done; the other five underwent pulmonary resections.

Though thoracoplasty during pregnancy has been referred to quite frequently in the medical literature,[1] in only two cases has pulmonary resection been reported, as far as we could determine.

Thompson and Bressler[2] discussed a case of pneumonectomy which was carried out because of extensive caseous pneumonic disease in a 29-year-old woman, six months pregnant. Convalescence was relatively uneventful. Cesarean section had been elected, but the premature delivery of a 5 pound, 1 ounce, normal male infant occurred one month prior to term. Fourteen months following surgery both mother and child were in good condition. The mother's sputum tests were negative. Streptomycin had not been administered due to fear of fetal damage.

Schaefer[3] refers to a 28-year-old patient with far-advanced disease on whom a right upper lobectomy was performed during the second month of gestation. She received streptomycin during the entire course of her pregnancy. Delivery was normal with the aid of low forceps.

The case histories of the ten patients undergoing major thoracic surgery at the Nova Scotia Sanatorium during pregnancy are given in this article; the five thoracoplasties are presented first, followed by the five cases of pulmonary resection.

CASE REPORTS

Case 1.—M. M. First admission of this patient extended from April 7, 1934, to April 27, 1935; she was admitted with caseous bronchopneumonic disease containing cavitation 1 by 2 cm. which extended from the apex to the third rib on the left. There was no demonstrable parenchymal lesion on the right. She also suffered from tuberculous laryngitis. Left-sided pneumothorax was combined with phrenic crush and the patient returned home to continue pneumothorax in Cape Breton. Her condition apparently was satisfactory with cavity closure in May, 1936. However, a cavity, 3 by 3½ cm., became visible in February, 1938, and the patient was readmitted to the Nova Scotia Sanatorium.

Second admission continued from March 21, 1938, to Oct. 20, 1938. The admission roentgenogram disclosed a small amount of exudative productive disease opposite the first interspace on the right. On the left there was now a cavity measuring 5 by 3½ cm., extending between the first and third ribs. The pneumothorax had fully re-expanded but the diaphragm was still markedly elevated; the larynx was normal on this occasion. Thoracoplasty was carried out in three stages, with the removal of eight ribs, and the procedure was completed

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on April 29, 1938. She was a primipara, 33 years of age, and was five months pregnant at the time. Postoperative convalescence was uneventful. Cesarean section was done on Sept. 11, 1938, under spinal anesthesia. A living child was delivered and there were no complications. A chest film taken on Nov. 23, 1950, was satisfactory, and twelve years following thoracoplasty this patient was leading a normal life.

CASE 2.—C. D., aged 26 years, was admitted Feb. 10, 1941, and discharged Dec. 6, 1941. The history dated back to 1935 when this patient failed to heed medical advice on the discovery of a parenchymal lesion and continued to lead a normal life. Four years later she complained of weakness and loss of energy. A chest x-ray film then revealed marked extension of disease and she was admitted to a tuberculosis unit in Cape Breton. Cesarean section was carried out there on Jan. 15, 1940; the patient was treated with bilateral pneumothorax. She was discharged to her home in May, 1940, but further extension of disease was revealed in a film of December, 1940. Admission to the Nova Scotia Sanatorium was advised. The admission film of Feb. 12, 1941, showed an irregular pneumothorax with 40 per cent collapse on the right side, but no cavity was seen. On the left there was exudative productive disease extending from the apex to the third rib, with a cavity measuring 2¾ by 1¾ cm. opposite the first rib and interspace. A left-sided thoracoplasty was carried out in three stages which were completed on April 8, 1941; seven ribs were removed. The patient was in the fifth month of gestation at this time. Convalescence was satisfactory and a cesarean section was performed on Aug. 22, 1941. There were no complications to mother or child. A film dated Dec. 3, 1941, disclosed 30 to 40 per cent pneumothorax on the right and a satisfactory collapse due to thoracoplasty on the left. Subsequent progress was satisfactory as evidenced by the fact that the patient was leading a normal life in September, 1950, nine years after the surgical procedure. At the same time an x-ray film dated Sept. 15, 1950, showed no evidence of reactivation.

CASE 3.—F. W., aged 28 years, was first admitted March 26, 1943, and remained until June 13, 1944. The history dated back to 1939 when this patient began to suffer from excessive fatigue and frequent colds. In August, 1940, she developed right-sided dry pleurisy following which a film was taken and tuberculosis disclosed. She was admitted to a Cape Breton unit in November, 1940, where bilateral pneumothorax was instituted. Admission film at the Nova Scotia Sanatorium, taken March 29, 1943, disclosed a bilateral pneumothorax with apparent closure of the cavity on the right, but a cavity 2 by 1¼ cm. was seen opposite the first and second ribs (inner zones) on the left. By May, 1943, the right lung had re-expanded completely. A three-stage, six-rib thoracoplasty was completed for the left lung on July 2, 1943, during the sixth month of gestation. This patient was a primipara. Severe cardiorespiratory collapse occurred immediately following the third stage of thoracoplasty, but otherwise postoperative convalescence was satisfactory. Delivery of a healthy infant took place with the aid of midforceps on Sept. 18, 1943. By January, 1944, the cavity remained apparent in the underlying lung and measured 2¼ by 1¼ cm. The patient was passed for a revision of the thoracoplasty. However, she left for her home on June 13, 1944, and failed to return as planned; the last film was taken here on April 27, 1944.

Subsequently she was re-admitted on Dec. 2, 1947, to a Cape Breton unit, and a film on Dec. 8, 1947, revealed bilateral extension of disease with cavities measuring 5 by 4 cm. on the right and 4 by 2 cm. on the left side. In March, 1948, the right upper lobe had become atelectatic while on the left there was no change. On May 7, 1949, she was re-admitted to the Nova Scotia Sanatorium where a film on May 10, 1949, revealed multiple cavitation in the right upper lobe as well as cavitation in the underlying lung on the left side. Pneumoperitoneum was initiated on July 4, 1949, and has been continued to the present date. Extensive courses of chemotherapy, including PAS, totaling 7,597 Gm., and 153 Gm. of streptomycin have been administered. Sputum, which was strongly positive on admission, has been negative on culture even since cessation of chemotherapy. Bronchograms revealed advanced bilateral bronchiectasis. The patient's general condition is excellent but, of course, the prognosis remains guarded.

CASE 4.—E. M. was a 23-year-old multiparous, Indian patient. She was admitted on April 9, 1952, and discharged against medical advice on Dec. 20, 1952. The admission film disclosed a linear fibroid lesion opposite the first interspace on the right, while there was a cavity measuring 1½ by 1 cm. within a fibrocaseous lesion extending between the apex and first interspace on the left side. A single stage, five-rib thoracoplasty was carried out on May 13, 1952, during the third month of gestation. Postoperative progress was uneventful. On Nov. 1, 1952, there was a normal delivery of a healthy child, and on December 3, her last film disclosed satisfactory results as far as can be determined at such an early date.

CASE 5.—E. N. was 21 years old. This multiparous patient was admitted on Aug. 30, 1952, and presented no complaints. Passing by the mobile x-ray unit near her home, she decided to have a chest roentgenogram taken, and it disclosed pulmonary tuberculosis. Sputum tests were positive. The admission film taken at the Nova Scotia Sanatorium on Sept. 3, 1952, disclosed multiple cavitation extending between the apex and second interspace on the right side. On the left side there was a disseminated nodular lesion extending from the apex to the fourth rib, with suggestive cavitation opposite the first rib. The preoperative film taken on October 27 revealed slight bilateral improvement. On Oct. 28 and Nov. 13, 1952, a two-stage thoracoplasty was carried out; three ribs were removed on each occasion. Postoperative convalescence was satisfactory. The patient was then at the sixth month of gestation. On Feb. 19, 1953, she gave birth to a normal male infant.

CASE 6.—M. J. This patient was 35 years old. Due to cough, sputum, and fatigue, a chest film was taken in October, 1948, and it disclosed pulmonary tuberculosis. Bed rest treatment was carried out at home until June, 1949. From June 23, 1949, to Nov. 18, 1950, she was treated at the Nova Scotia Sanatorium. She was multiparous and her last menstrual period had taken place on June 1, 1949. The admission chest film disclosed on the right, scattered productive disease extending between the apex and fourth interspace, while on the left, the upper lobe was partially atelectatic and mainly productive disease extended from the first interspace to the base. Bronchoscopy was carried out to determine the cause of the atelectasis on July 25, 1949, and a type II[1] tracheobronchial lesion involving the left main bronchus was disclosed. A total of 60 Gm. of streptomycin was administered between July 26 and Sept. 21, 1949. No ulceration was apparent in the follow-up bronchoscopic examination on Aug. 28, 1949. A diagnosis of pregnancy was made on Aug. 1, 1949, and at the time of her apical posterior segmental resection in October, the patient had reached the fourth month of gestation. Streptomycin (37 Gm.) was administered between Oct. 11 and Nov. 16, 1949, making a total of 97 Gm. during pregnancy. Pathologic examination of the resected segment revealed fibrocaseous tuberculosis without cavitation. Convalescence was satisfactory, and on March 8, 1950, a normal delivery took place.

As sputum examinations continued to be positive, a further bronchoscopic examination was carried out on April 21, 1950. This disclosed a recurrence of the tracheobronchial disease with a shallow ulcer involving the left main bronchus. Bronchoscopic examinations were carried out at monthly intervals until normal findings were finally revealed on Aug. 18, 1950; a further course of streptomycin had been given between April 29, 1950, and Aug. 10, 1950, totaling 56 Gm. of streptomycin and 47 Gm. of dihydrostreptomycin with PAS, 10 to 12 Gm. daily. Subsequent progress was uneventful; the final film was that of Oct. 14, 1950.

CASE 7.—E. B. This 23-year-old, multiparous woman was admitted Feb. 8, 1949, and discharged July 15, 1950. The condition was originally diagnosed in July, 1947, when a chest film was taken because of fatigue and loss of strength. She was treated by bed rest at home until November, 1947, following which she recommenced work. In January, 1949, however, admission to the Sanatorium was advised. The admission film disclosed a normal right lung and exudative disease on the left side extending from the third rib to the base. However, no cavity was present although the sputum was strongly positive. Bronchoscopic examination was carried out to rule out tracheobronchitis as the source of the positive sputum. This procedure revealed ulceration and exudate situated in the left main bronchus.

Further bronchoscopic examination on May 28, 1949, after chemotherapy, revealed complete healing of the tracheobronchial disease. Chest film on June 20, 1949, showed improvement, while a bronchogram done on July 7, 1949, revealed considerable bronchiectasis, both in the lingular division of the upper lobe and also throughout the lower lobe. Resection was carried out on Sept. 30, 1949. A total pneumonectomy was required due to the presence of a hard mass in the anterior segment of the upper lobe in addition to the bronchiectatic disease. At this time the patient was about eight weeks pregnant. Convalescence was uneventful and on April 24, 1950, she was delivered by the normal route. She was discharged two months later in July, 1950, and subsequent progress has been satisfactory; the last film seen at the Nova Scotia Sanatorium was taken Feb. 20, 1951.

CASE 8.—S. M. was 19 years old. This primiparous patient was first admitted on Jan. 8, 1948, and was discharged on May 8, 1949. A film in October, 1947, was taken because of cough. This disclosed pulmonary tuberculosis and sputum tests were positive. Admission chest film revealed an advanced, mixed, exudative, productive lesion extending between the apex and third rib on the right with cavity measuring 1 by 1 cm., while the left lung was negative. Right initial pneumothorax was performed on Feb. 6, 1948, and a successful pneumolysis was carried out on April 7. She was discharged in May to continue pneumothorax at home.

Following re-expansion in March, 1951, there was a recurrence of cough and positive sputum. She was therefore readmitted to the Sanatorium on Aug. 13, 1951, where she remained until Feb. 17, 1952. Readmission film disclosed extension of disease with a 10 per cent collapse due to artificial pneumothorax on the right. A rounded lesion was seen in the upper lobe. The left lung was negative. Bronchoscopic examination on September 13 revealed no tracheobronchial disease. Right upper lobectomy was carried out on Oct. 12, 1951.

Pathologic report of the resected lobe revealed an area of caseation measuring 4 by 3 cm. Streptomycin was administered, 1 Gm. daily, between Oct. 8 and Nov. 11, 1951, and PAS was given to a total of 660 Gm. Pregnancy had not been diagnosed prior to the resection, but on Feb. 8, 1951, examination revealed the presence of a seven-month fetus. The patient was therefore approximately three months pregnant at the time of resection. Subsequent progress has been satisfactory with a normal delivery occurring following discharge, which she took against advice because of her pregnancy.

CASE 9.—L. F. This multiparous, 30-year-old patient was admitted March 13, 1950, and discharged June 30, 1952. Disease was originally diagnosed in October, 1949. Admission film revealed a cavity measuring 2 by 2 cm. opposite the first interspace on the left side. The right lung was normal. The patient was inoperable for left pneumothorax and therefore a six-rib thoracoplasty was carried out. This was completed on May 4, 1950.

In June, 1951, sputum was still positive on culture. Planigraphic series revealed no cavity in the underlying lobe, but a bronchogram showed bronchiectasis involving the upper lobe. Left upper lobectomy was carried out on Nov. 6, 1951, at which time the patient was four months pregnant. Postoperative convalescence was satisfactory, and there was a normal delivery on April 7, 1952. Since discharge on June 30, 1952, follow-up films have proved satisfactory; the last was taken on Nov. 19, 1952. She has no positive sputum.

CASE 10.—M. D. Commencing on Oct. 1, 1948, this 22-year-old primiparous patient had undergone three periods of treatment at one of the tuberculosis units, where a 50 Gm. course of streptomycin with PAS was given prior to transfer to the Nova Scotia Sanatorium on May 23. The admission chest film revealed an advanced caseous process containing a large thick-walled cavity and extending from the apex to the second rib on the right side. A film taken July 12, 1952, revealed considerable extension in the same lobe. Bronchoscopic examination was satisfactory, and on July 15, 1952, a right upper lobectomy was performed. The patient's last menstrual period was that of Dec. 28, 1951, and she was therefore in the sixth month of gestation. The immediate postoperative course was stormy. Her temperature, which had been 99° F., rose to 104° F. on the first postoperative day. There was also marked nausea and vomiting.

On the second postoperative day labor commenced, and the patient was delivered of a premature infant weighing 3 pounds, 2 ounces. Two days later the temperature fell to its preoperative level. The baby died of prematurity on the third day. Postoperative convalescence continued to be rather stormy, and the film of July 22, 1952, revealed marked contralateral spread of disease. Following this, however, there has been steady clinical and radiologic improvement. The latest roentgenogram was dated December 10, five months following the surgical procedure.

DISCUSSION

In recent years it has been the general consensus that the prognosis of the tuberculous pregnant woman is little affected by her pregnancy, provided adequate treatment is given during and following her pregnancy. This fact was borne out in our previously reported series of sixty-six cases. Among the fifteen patients in the far-advanced group treated during their pregnancies and delivered at the Sanatorium, only two died. Of six patients in similar circumstances, who were discharged prior to term, none survived.

It is felt, also, that resectional surgery should be offered to the pregnant woman as well as to the nonpregnant, particularly in the presence of extending disease of caseous pneumonic or fibrocaseous character. Of the first 250 pulmonary resections carried out at the Sanatorium, five have been performed on pregnant women. The choice of this form of surgery might be criticized very late in pregnancy as in Case 10, where a premature delivery with the subsequent death of the infant resulted. Probably in retrospect, we should have waited an additional month to ensure viability of the fetus, but in that same month, disease already known to be extending might have reached the point where surgery was impossible either then or later. In the other four cases of resection, the operative procedure had no effect on the fetus. One operation was done during the third month of gestation in a woman not known to be pregnant; one was carried out during the second month; and two during the fourth month of pregnancy.

TABLE I. TYPE OF OPERATION RELATED TO PARITY AND MONTH OF GESTATION

THORACOPLASTY				RESECTION			
CASE NO.	RIBS	PARITY	MO. OF GESTATION	CASE NO.	TYPE OF RESECTION	PARITY	MO. OF GESTATION
1	8	P	5	6	Seg.	M	4
2	7	M	5	7	Pneum.	M	2
3	6	P	6	8	Lob.	P	3
4	5	M	3	9	Lob.	M	4
5	6	M	6	10	Lob.	P	6

P = Primipara.
M = Multipara.

The five thoracoplasties, which with one exception were multiple stage procedures, exerted no adverse effect on the infants. Two of these were done during the sixth month, two during the fifth, and one at the third month of gestation.

It is now felt that streptomycin, which is so indispensable in resectional surgery, can be administered with safety from the point of view of possible fetal damage. Though it has been determined that the level of this drug in

the fetal blood reaches one-half that in the maternal blood stream, there have as yet been no reports of damage to the eighth nerve of the fetus, nor has there been in any of this series to date.

SUMMARY

1. The histories of ten persons having had major chest surgery performed during pregnancy have been presented. Five patients were subjected to thoracoplasty and five to pulmonary resection.

2. It would appear that major collapse therapy or resectional surgery when indicated should be offered to the pregnant just as to the nonpregnant tuberculous woman.

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Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 34th annual meeting of the American Association for Thoracic Surgery will be held May 3, 4, and 5, 1954, in Montreal, Que., Canada. Headquarters will be at the Sheraton-Mount Royal Hotel.

Requests for Hotel Reservations

These should be mailed *directly* to the Reservation Manager, Sheraton-Mount Royal Hotel, Montreal, Que., Canada. Please mention this Association, type of accommodation desired, date, and approximate hour of arrival. If accommodations are desired elsewhere, please communicate directly with hotel of your choice.

Thoracic Surgical Forum

After due consideration, the Council has decided to try an experiment by assigning one morning session to a "Thoracic Surgical Forum" for the presentation of short papers on current thoracic research, anatomy, modified surgical techniques, etc. Papers at this session will be limited sharply to ten minutes (including the showing of lantern slides or other illustrative material) with three minutes as the maximum discussion of each paper.

These papers will be published together in one issue of THE JOURNAL OF THORACIC SURGERY. Maximum length: 3,000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts for Papers

Abstracts for the presentation of papers at the 1954 meeting, including papers before the Forum, must be received on or before January 1, 1954, otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Forum" or "For Regular Program."

Five, repeat five, copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words and accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee not later than Jan. 1, 1954, to be considered for action in Montreal. Applications received beyond this deadline will be deferred for consideration until 1955.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the sponsoring statement on the application form, the sponsors *must* submit a separate letter concerning the applicant direct to Dr. Lyman H. Brewer, III, Chairman of the Membership Committee, 2010 Wilshire Blvd., Los Angeles, Calif. Completed application forms should likewise be sent to Dr. Brewer. These regulations are at the direction of the Council and were made effective in 1952 at Dallas.

Correspondence

To the Editor:

Some time ago, my attention was called to an article by Leslie J. Temple, F.R.C.S., of Liverpool, England, which appeared in The Journal of Thoracic Surgery 19: 142 (March) 1950. The article is entitled, "Aneurysm of the First Part of the Left Subclavian Artery." In his report, the author states that during the course of an operation for subclavian aneurysm, he performed transpleural ligation of the first portion of the left subclavian artery. He also mentions an unpublished case of Mr. H. Moriston Davies in which a similar procedure was performed in June, 1944. At one point, Mr. Temple states as follows: "As I have pointed out, apart from Moriston Davies' unpublished case, I know of no direct transpleural approach to the left subclavian artery."

For the benefit of those who may wish to collect a set of references dealing with intrapleural ligation of the first portion of the left subclavian artery, may I call attention to an article of mine entitled, "Transpleural Transthoracic Ligation of the First Portion of the Left Subclavian Artery" which appeared in *Surgery* 10: 747 (November) 1941. In my article, transpleural ligation of the first portion of the left subclavian artery was performed as a preliminary to mid-humeral amputation, in a case of congenital, multiple, arteriovenous communications involving the left upper extremity. To the best of my knowledge, my case was the first in which the basic principle of the intrapleural subclavian artery ligation was performed.

Arthur S. W. Touroff, M.D.